QUANTITATIVE GENETICS OF *PUCCINIA SORGHI* RESISTANCE AND HUSK NUMBER IN *ZEA MAYS*

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By

Soon Kwon Kim

Dissertation Committee:

James L. Brewbaker, Chairman
Ivan W. Buddenhagen
Peter P. Rotar
James C. Gilbert

We certify that we have read this dissertation and that in our opinion it is satisfactory in scope and quality as a dissertation for the degree of Doctor of Philosophy in Horticulture.

DISSERTATION COMMITTEE

James L. Brewbaker
Chairman

J Buddenhagen

Peter P. Rotar

James C. Gilbert

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INTRODUCTION

Plant diseases have been among the principal hazards affecting crop productivity since man began to cultivate crops. Development of resistant crops is one of the most dramatic and the best known contributions of plant breeding toward an adequate supply of food and feed for human beings and animals.

Corn (Zea mays L.) is among the three most important cereal crops in the world, including wheat and rice. It is grown throughout the temperate, subtropical, and tropical regions wherever rainfall or irrigation temperature, and sunlight are adequate.

Puccinia spp. rusts probably rank first in economic importance among cereal diseases (Hooker 1967). Three rust species are known to occur on maize, Puccinia sorghi Schw., Puccinia polysora Underw. and Physopella zeae (Mains) Cumm and Ramachar (Ullstrup 1955, Wood and Lipscomb 1956, Hooker 1962, and Schieber and Dickson 1963). The latter two diseases have not been identified conclusively in Hawaii (Brewbaker et al. 1972). Common corn rust caused by P. sorghi is widely distributed throughout the world wherever corn is grown (Arthur 1934, Wood and Lipscomb 1956, and Hooker 1969). P. sorghi thrives under moderate temperatures and high humidity levels. Such environmental conditions generally prevail in the winter corn nurseries on the Waimanalo Farm, where the rust has been epiphytotic since 1968 (Brewbaker et al. 1972).

The loss of corn grain yield due to P. sorghi is rarely of major economic importance in North America and many other regions. Many of the commercially important field corn hybrids in the American Corn Belt are moderately resistant to the rust in the mature plant stage (Ullstrup 1955,

Russell and Hooker 1959, Hooker 1962, Russell 1965). Results of our corn yield trials to be reported, however, indicate that losses can be substantial in Hawaii. No Hawaiian varieties or lines of corn are resistant to P. sorghi. Many of the widely used corn inbred lines in the U. S. Corn Belt and tropical regions also are severely attacked under our environmental conditions in Hawaii. Therefore it is important to study maize under severe P. sorghi epiphytotic pressure in order to identify effective sources of resistance to the rust.

The corn earworm, Heliothis zeae (Boddie), is widely distributed throughout the world and limits the economic production of sweet corn as well as field corn (Painter 1968). Resistance to the corn earworm is known to be complex (Widstrom 1967), involving husk length, tightness of husks, balling of silks and tassel-tipping of ear. Very few studies has been made, however, on the inheritance of corn husk number, which may be of significance in corn earworm resistance (Collins and Kempton 1917, McClelland 1929, Poole 1940, Painter 1968).

The investigations to be reported were made to assess the rust resistance of widely used maize inbreds and races, to assess the effect of rust on yield in Hawaii, to study host-pathogen interactions, and to determine gene action controlling generalized rust resistance and husk number in maize.

LITERATURE REVIEWS

Genetic Control of Disease Resistance

The earliest evidence for genetic systems controlling disease resistance came from studies of the yellow wheat rust caused by *Puccinia glumarum*. Biffen (1905) found that resistance was controlled by a single recessive gene. Dickson (1950), and Hooker (1967) have reviewed the genetics of disease resistance in cereals, and Walker (1965) reviewed the genetics of resistance in vegetable crops. Stevenson and Jones (1953) listed the sources of resistance in crop plants in a Yearbook of the United States Department of Agriculture devoted to plant diseases.

Flor (1955) found gene for gene relationships in flax and flax rust, *Melampsora lini.* For each gene conditioning race specific resistance in the host, Flor established that there is often a specific and related gene in the pathogen that conditions the ability to overcome that resistance. The gene-for-gene theory has been supported by Moseman (1958) in *Hordeum vulgare* and in *Erysiphe graminis*, by Powers (1960) for powdery mildew, and by Oart (1963) for *Ustilago tritici* of wheat. Person (1969, 1962) has proposed that the gene-for-gene relationship may occur as a general rule for host : parasite systems.

Coons (1953), Reits (1954), Stakman and Harrar (1957), Walker (1959), and Nelson (1973) have discussed the economic value of breeding for disease resistance. Coons (1953) estimated about 50 percent of the crop acreage in the United States utilized resistant varieties. This had increased to more than 75 percent of U. S. agricultural acreage by 1972 (Nelson 1973).

Mycology, Taxonomy, and Host Range of P. sorghi

P. sorghi is a macrocyclic heteroecious fungus. Aecial and pycnial stages of the rust are known in corn (Stakman 1940, Ullstrup 1955, Hooker 1973). Mycelia grow primarily in the chlorenchyma (Mains 1917), with appressoria formed over the stomato and penetration pegs that enter through them (Weber 1921). The initial stages of infection are similar in both resistant and susceptible reactions (Wellensiek 1927). No cytoplasmic reactions were observed beyond an initial reduction in chlorophyll formation at the infection site (Flangas and Dickson 1961).

Hilu (1965) studied in detail the host-pathogen relationships of P. sorghi in resistant and susceptible corn seedlings. Resistance or susceptibility appeared as different reaction types, but initial stages of infection were identical in both resistant and susceptible seedlings. Hilu described the initial stages of infection as urediospore germination, appressorial formation, stomatal penetration, vesicle formation, intercellular hyphal growth in the mesophyll, and intracellular haustorial development. Hilu and Hooker (1967) observed that the infection types can be modified by air temperature, light intensity and duration. Van Dyke and Hooker (1969) described the ultrastructure of host and pathogen in interaction of corn with P. sorghi, and noted its similarity to that of many other fungal diseases. The rust fungus displayed vacuoles, storage bodies, occasional unidentifiable inclusions and a limited endoplasmic reticulum, but had no recognizable Golgi apparatus.

P. sorghi usually infects leaves, where it produces a round urediospore. Severe rust has also been observed to infect on the leaf blade, leaf sheath, husk, and tassel (Stakman et al. 1929, Arndt and Stevenson 1946). However, P. sorghi less frequently infects the husks and

the floral bracts of the tassel (Dickson 1956).

Corn rust spores are transmitted by air or wind (LeRoux and Dickson 1957, Hooker 1969), by seeds (Rhind 1952, Reyes 1953) and probably also by infected or contaminated leaf material (Laundon and Waterston 1964).

Alternate hosts are known to be several Oxalis species, chiefly O. corniculata L., O. europaea Jord (O. Cymosa Small), and O. stricta L. (Dickson 1956, Flangas and Dickson 1961). However, infection of alternate host has not been reported in Hawaii.

Geographical Distribution of P. sorghi

Three species of corn rust are known to occur on maize. They are P. sorghi Schw., P. Polysora Underw., and Angiospore zeae or Physopella zeae (Mains) Cumm and Ramachar (Hooker 1962b, Schieber and Dickson 1963). Common corn rust, caused by P. sorghi is distributed wherever corn is grown. Ullstrup (1955) noted that urediospores of P. sorghi may overwinter in southern and central United States and produce infections on maize in the following spring. But Hooker (1969) clearly indicated that P. sorghi does not over winter in the Corn Belt.

Southern corn rust, caused by P. polysora, is distributed in the warmer, more tropical areas of the world (Ullstrup 1955, Laundon and Waterston 1964, Hooker 1973). Southern corn rust was particularly severe in parts of West Africa from 1949 to 1951 and caused substantial yield losses (Rhind 1952, Rhind et al. 1952, Nattrass 1952, Nigeria Agri. Dept. 1953, Cammack 1954, Ullstrup 1955, Hooker 1962b, Van der Plank 1968). P. polysora is also known to occur in the United States (Ullstrup 1955, 1958, 1965). Tropical rust, caused by Angiospore zeae or Physopella zeae, does not occur in the United States but is present in Central America,

Mexico, and parts of South America (Cumins 1941, Muller 1953, Robert 1962, Hooker 1962, Grant et al. 1963).

It is very hard to distinguish P. sorghi from P. polysora. However, P. sorghi has been distinguished from P. polysora by the larger, usually more elongated and sparse uredia; the regular, well rounded, darker urediospores; the black open telia, and the well rounded teliospores, with thicker apical walls and long pedicels (Mains 1938, Stakman 1940, Cumin 1941, Wood and Lipscomb 1956, Laundon and Waterston 1964). Resistance to sorghi does not confer resistance to P. polysora, and vice versa. Hence distinguishing between P. sorghi and P. polysora based on host reactions of the maize lines to the rusts has been recognized as a more reliable approach. Cumin (1941) reported that A. zeae resembles P. polysora somewhat in the uredial stage and has been often confused with it, but P. polysora has hyaline urediospores and covered telia containing sessile teliospores.

Effect of Rust on Corn Yield

Stakman and Christensen (1926) and Stakman et al. (1928) expressed concern that P. sorghi might become an important disease and destroy newly developed lines of maize. Unusually severe P. sorghi epiphytotics have been reported from several states in the U. S. such as in South Carolina by Arndt and Stevenson (1946) Virginia by Roane (1950), Iowa by Vestal and Semeniuk (1951), Florida by Townsend (1951), and Wisconsin by Dickson (1952). Wallin (1951) summarized that yield losses due to P. sorghi were not significant in the main Corn Belt states, but because of the importance of corn, rust was common enough increase to arouse concern. In Kansas, greater loss was considered due to high morning and afternoon humidity

in July. Wallin cautioned that P. sorghi constituted a continuing potential threat in the Corn Belt. More recently, the potential significance of yield losses due to P. sorghi also have been reported in the Corn Belt (Hooker 1962, Russell 1965).

Hooker (1962) reported a 6.3% grain yield reduction due to rust inoculations when susceptible hybrid (B14 X Oh41) was compared with its resistant counterpart (B14A X Oh41) in inoculated plots. Differences were highly significant and 30.4% rust coverage of the leaf surfaces estimated. Little rust developed in uninoculated plots under natural rust infections, where the yields of susceptible and resistant hybrids were essentially identical.

Russell (1965) observed a 19.8% grain yield reduction due to rust when three susceptible hybrids were compared with three resistant counterparts in inoculated plots. Differences were highly significant in grain yield and grain moisture in inoculated plots and a maximum 70% rust coverage of the leaf surfaces estimated. The greatest yield reductions were observed in crosses involving susceptible inbred Oh7K. The average grain yield reduction under natural infections was 3.9% in the uninoculated plots and this difference was significant. Resistant hybrids had significantly higher grain moisture than their susceptible counterparts in the inoculated plot. Russell recommended that resistant inbred B14A should replace highly susceptible counterpart B14 in commercial use because, if more extensive P. sorghi development occurred, substantial yield reduction might be prevented.

P. sorghi is also common in the Philippines (Reyes 1953), Kenya (Nattrass 1952), and Nepal (Manandhar and Shah 1972), causing considerable reduction in yield.

The failure of corn rusts to produce severe epiphytotics in the tropics is considered by Borlaug (1965) to be the result of the heterogeneous maize populations, such as synthetics or open-pollinated varieties. Borlaug observed P. sorghi attacking much more severely on double-cross hybrids than open-pollinated varieties.

The following study was initiated to assess the effect of rust on grain yield and other agricultural characteristics in maize under severe natural P. sorghi epiphytotic pressures prevailing in Hawaii.

Inheritance of Rust Resistance

The resistance in maize to P. sorghi includes specific and generalized resistance. The latter has also been called general resistance or nonspecific resistance (Hooker 1967, 1969).

Specific resistance, based upon host hypersensitivity, is qualitative in expression and is definitely race specific (Hooker 1969). This type of resistance is expressed through the plant stages in the form of chlorotic or necrotic leaf flecks, or small pustules surrounded by chlorotic or necrotic tissue. Inheritance of specific resistance to P. sorghi Schw. has been elucidated especially by Hooker and his colleagues (Mains 1931, 1962; Russell and Hooker 1959; Hooker 1962, 1963, 1967, 1969; Hooker et al. 1955; Hooker and Russell 1962; Malm and Hooker 1962; Lee et al. 1963; Hagan and Hooker 1965; Hooker and Saxena 1967, 1971; Wilkinson and Hooker 1968).

Main (1931) was the first to report that specific resistance to rust caused by P. sorghi is controlled by a single dominant gene. Rhoades (1935) located the rust resistance gene in corn, on the distal one-fourth of the short arm of chromosome 10 through a cytological studies of X-ray induced deficiencies and genetical studies of trisomic ratios. This gene,

in line GG208R, was first designated Rp (Rhoades and Rhoades (1939), later as Rp_1 (Russell and Hooker 1959); the allele was later designated Rp_1^a (Hooker 1963). The location has been confirmed by Russell and Hooker (1962). Other rust resistance genes Rp_5 and Rp_6 also occur on chromosome. Gene Rp_5, first identified in corn line Uruguay 191, is lined to Rp_1 by about 1.1 ± 0.2 crossover units. Gene Rp_6, in corn line Turkey 597, is also linked to Rp_1 by about 2.1 ± 0.4 crossover units. Rp_1 is located between Rp_5 and Rp_6 (Hagan and Hooker 1965, Wilkinson and Hooker 1968). In addition to these three Rp genes, P. polysora resistance gene, Rpp_9 also occurs on chromosome 10 and is linked to Rp_1^d by about 1.6 crossover units (Ullstrup 1965). Russell and Hooker (1962) demonstrated that other dominant genes for resistance to P. sorghi also occur on chromosomes 3 and 4, and were designated Rp_3 and Rp_4 respectively. Van Dyke and Hooker (1969) reported that gene Rp_4 in Ethiopian corn line 906 showed a "Z" reaction to P. sorghi. In this type, the reaction consists of resistant flecking of the tips of leaves, while chlorotic lesions are susceptible. So far more than 100 alleles or sources of specific resistance to P. sorghi have been reported by testing inbred lines and open-pollinated varieties (Hooker et al. 1955; Hooker and LeRoux 1957; and Hooker 1962, 1969). Among the specific resistance genes, Rp_1^d and Rp_1^g appear to be the most effective on a world wide basis in conferring hypersensitivity at the present time (Hooker 1969).

In addition to specific dominant genes, recessive genes for resistance to P. sorghi have also been reported. In the resistant inbreds Amarge 47 and Midland 125, high resistance was due to the multiple recessive condition of genes rpa, rpb, and rpc (Malm and Hooker 1962; Hooker 1962; Hooker 1969). The presence of modifying genes for resistance to P. sorghi

also has been studied in several ways (Lee et al. 1963; Malm and Hooker 1962; Wilkinson and Hooker 1968; Hooker 1969). Even though the importance of modifying genes is not clear at this time, future studies of corn rust may be of value in this respect.

Generalized resistance is quantitative in expression and is clearly race non-specific (Hooker 1969, Nelson 1973). This type of resistance is usually expressed in mature plant stages and ranges in a continuous series from high resistance to moderate resistance. In general, highly resistant plants have only a few pustules on the leaves at mature plant stage while highly susceptible plants have numerous pustules (Hooker 1973). Very few studies have been carried out on the genetics of generalized resistance to P. sorghi in maize. Studies on generalized rust resistance in o.her cereals are also somewhat limited (Caldwell 1966, Chester 1946, Hooker 1962, and Murray 1961). However, the importance of the generalized resistance in crops to diseases has been long recognized by the plant breeder and plant pathologist and thoroughly reviewed by Van der Plank (1968) and Nelson (1973).

Hooker (1962, 1967a, 1967b) studied the inheritance of generalized resistance in maize to P. sorghi in Illinois. Approximately 10 plants of each nonsegregating parental and F_1 progeny, and 100 plants of each segregating population from 64 crosses were scored individually on the basis of percentage of leaf area infected, using the rating scale of Peterson et al. (1948). Heritability estimates were obtained by using variation among plants of the nonsegregating progenies as an estimate of environmental variance. In 45 of the 65 crosses the heritability estimates were over 85%. Hooker did not study backcross progenies, which should estimate heritability more accurately. It should not be difficult in a

breeding program to select for generalized resistance. Hooker (1969) reported that a large proportion of the U. S. inbred lines were highly resistant to P. sorghi. A testing program for generalized resistance to P. sorghi was suggested as a part of maize breeding programs.

In sharp contrast, it is clear that most of the widely used maize inbred lines are highly susceptible under the severe rust epiphytotic pressure prevalent in Hawaii. For example, inbreds 38-11 and C103 were highly resistant in Illinois (Hooker 1962), but they were moderately or highly susceptible, respectively, in Hawaii (Table 3). P. sorghi epiphytotics in Illinois were not as severe as epiphytotics in Hawaii. It is important to study genetics of generalized resistance under severe P. sorghi epiphytotic pressure in order to determine more accurately gene actions conditioning generalized resistance in maize.

Host-Pathogen Relationship

Quantitative changes in peroxidase activity has been observed to be induced by pathogenic infection, by senescence, and by wounding, and may be correlated with resistance of the host to a pathogen (Farkas and Stahmann 1966; Jennings et al. 1969; Peirce and Brewbaker 1973). Rubin et al. (1947) observed that oxidase conversions of phenols in potato were associated with resistance to Phytophthora infestans. Later Fehrmann and Diamond (1967) and Lovrekovich (1967) found that the level of peroxidase activity in potato leaves before infection was positively correlated with resistance to P. infestans.

In sweet potato, Loebenstein and Linsey (1961) demonstrated that virus infected leaf and root tissues had significantly more peroxidase activity than their healthy counterparts, and this increase in the

peroxidase activity was associated with the symptom appearance on the tissues. In tobacco, Yu and Hampton (1964) found that tobacco leaf tissue infected with Colletotrichum destructivum showed higher peroxidase activity than the healthy counterpart. Loebenstein and Linsey (1966) also found that tobacco leaves severely infected with mosaic virus showed the highest peroxidase activity. Lovrekovich et al. (1968) obtained a correlation between the level of peroxidase activity in tobacco leaves and resistance to Phytophthora tabaci, this correlation associated with the aging of the leaves. In flax, Andreev and Shaw (1965) found that infected leaf tissue with Melampsora lini rust had more peroxidase isozymes than its healthy counterpart.

In beans, Farkas and Stahmann (1966) found that virus infected bean leaf tissues had more peroxidase isozymes than healthy leaf tissue. At least four different peroxidases I, II, III and IV were isolated from the virus infected tissues. Peroxidases II and III did not exist in healthy tissues. Recently, Novacky and Hampton (1968) demonstrated that peroxidase activity in several virus infected plants were changed quantitatively by both infection and by senescence. These changes, however, were not identical. They used Nicotiana tabacum infected with tobacco mosaic virus, Vigna sinensis infected with cucumber mosaic virus, P. vulgaris infected with Southern bean mosaic virus.

In maize, Jennings et al. (1969) reported that increased activity of peroxidase and polyphenoloxidase was associated with Helminthosporium carbonum infection. Resistant plants infected with H. carbonum races showed higher peroxidase activity than healthy counterparts, but did not show differences in polyphenoloxidase activity. The infected susceptible plant also showed higher peroxidase activity, and polyphenoloxidase

activity showed a similar response to peroxidase. They concluded that increased peroxidase and polyphenoloxidase activities were related to susceptibility in the host.

Pryor (1971) has studied phenol oxidase of corn infected with P. sorghi rust. He proposed a biochemical system which contained all the requirements of the hypersensitive response observed in resistant corn plants containing monogenic Rp genes. The model provided the necessary specificity, resistant host range, and virulence genes which would be easily detectable from the biochemical approach.

Hare (1966) reviewed the hypersensitivity form of resistance in cereals. Resistance to Puccinia spp. in cereals and some other diseases involves a hypersensitive response in which the host cells is killed and give a white fleck type hypersensitivity to resistant plant. Thatcher (1943) stated that the hypersensitive type of resistance might result from an extreme susceptibility to the pathogen, resulting in local lesions which prevent further sporulation of the pathogen inoculum.

Many phytoalexins produced in response to pathogenic and non-pathogenic organisms in crops have been reported. In Glycine max Klarman and Gerdemann (1963) found a phytoalexin produced in response to Phytophthora megasperma var. sojae. When they inoculated the H and H63 soybean varieties with P. sojae, extracts from the infected resistant H63 plants showed toxicity to this pathogen, but extracts from infected susceptible H plants or uninoculated plants did not show toxicity to P. sojae. They concluded that the ability of resistant soybean plants to produce the phytoalexin did not depend on the presence of the single dominant gene which conferred resistance on variety H63. Frank and Paxton (1970) found that resistant H63 and susceptible soybean varieties

produced phytoalexin equally within the first four hours after infection, but the susceptible H variety did not produce the phytoalexin after the four hours. They concluded that the resistance to P. sojae was related directly to the presence of phytoalexin in soybean plant.

In Zea mays, Lim et al. (1969, 1970) found that the resistance to Northern corn leaf blight caused by Helminthosporium turcicum Pass. was due to production of phytoalexins in the resistant host. Their results indicated some similarity with the phytoalexin production in soybean plant infected with P. sojae. They attempted to induce phytoalexin in resistant and susceptible maize with H. turcicum pathogen isolated from corn ("corn isolate") and with H. turcicum non-pathogenic to maize isolated from Johnson grass ("Johnson grass isolate"). The corn isolate induced two phytoalexins, A_1 and A_2 in the monogenic resistant maize containing Ht gene, but the Johnson grass isolate did not stimulate production of the phytoalexins. The phytoalexin production in maize has been limited to plants containing a monogenic resistance gene (Ht).

Hooker (1967) pointed out that no common hypersensitive forms of resistance to obligate pathogens such as P. sorghi in maize have been explained in terms of phytoalexins. Wingard (1953) also discussed defense mechanisms developing or induced in the host plant as the "active type" in response to pathogen infection. He classified nutritional defense to be of the "passive type", because the defense mechanism is already present in the host prior to pathogen infection. Hooker (1967) and Wood (1967) reviewed the physiology of disease resistance, noting several defense mechanisms operative in the host plant. Some mechanisms were active before infection ("passive type"), and others active after infection (active type"), the former expressed in the form of nutritional, chemical,

and physical states. For example, Link et al. (1929) found that the pigmented onion bulb producing protocatechuic acid conferred the resistance to onion smudge, caused by Colletotrichum circinans.

The objectives of the following study were to assay peroxidase activity of healthy and inoculated plants and to determine gene actions controlling peroxidase activity upon Puccinia sorghi infection in maize.

I. EFFECT OF RUST ON CORN YIELD

ABSTRACT

Ten paired double-cross hybrids, genetically similar except for the gene Rp_1^d, conferring resistance to Puccinia sorghi Schw., were studied under severe natural rust epiphytotics. Tests were conducted at the Waimanalo Farm in summer and in winter. Nine agronomic characteristics were measured in the summer trial, including grain yield, total fresh plant weight with and without ear, 1000 kernel weight, plant height, ear height, and days to mid-silk. All characteristics except for ear and plant height were reduced significantly due to rust at the 1% level. For example, grain yields at 15.0% moisture were reduced from 118.0 q/ha to 86.4 q/ha due to rust, with an average of 26.6% reduction. Total fresh plant weights at harvest were reduced from 493.9 q/ha to 366.7 q/ha, with an average of 25.8% reduction. Significant differences among 10 hybrids were detected for all characteristics except for grain yield and ear diameter.

Thirteen characteristics were measured in the winter trial, including dry plant weight without ear, kernel diameter, kernel width, and kernel depth in addition to the nine characteristics measured in summer. All characteristics except for kernel width were reduced significantly due to rust at the 1% level. For examples, grain yields at 15.0% moisture were reduced from 36.9 q/ha to 20.9 q/ha, with an average of 42.8% reduction. Total fresh plant weights at harvest were reduced from 257.0 q/ha to 185.9 q/ha, with an average of 27.4% reduction. Significant differences among 10 hybrids for all characteristics except for three kernel dimensions were detected.

Data combined over the two seasons indicated that the rust affected all nine characteristics significantly at the 1% level. Average percent reductions due to rust were 30.8% (grain yield), 26.5% (total fresh plant weight with ear), 24.1% (fresh plant weight without ear), 9.9% (1000 kernel weight), 11.0% (ear length), 9.5% (ear diameter), 3.4% (plant height), 4.6% (ear height), and 1.4 days (mid-silking). Highly significant treatment X seasonal interactions were detected in grain yield, total fresh weight with and without ear, ear diameter, and days to mid-silk. Highly significant differences among the 10 hybrids were detected for all characteristics except for grain yield and 1000 kernel weight. The significant yield reductions obtained in this study give an indication of the potential damage that may be caused by P. sorghi in maize in subtropics as well as temperate regions.

MATERIALS AND METHODS

Ten double-cross hybrids (Table 1) involving 11 parents were planted in paired rows at the Waimanalo Farm, University of Hawaii, April 1973 ("summer") and in November 1973 ("winter"). These paired rows were segregating for the monogenic rust resistance gene, Rp_1^d, and resistant plants were left in one row and susceptible plants in the other. Rp_1^d was derived from the Peruvian variety Cuzco. It was backcrossed into the B14 inbred; the resistant conversion was named B14A and released by Iowa State University in 1962 (Russell 1965). The Hawaiian inbred AA25 is a conversion of B14A to the Mv gene for resistance to Maize Mosaic Virus I (MMVI) through backcrossing in Hawaii. Rp_1^d gene is located on the short arm of chromosome 10 in maize and is completely dominant (Russell and Hooker 1962, Russell 1965, Hooker 1962, 1969).

The ten following doublecross hybrids were used:

Table 1. Pedigree of doublecross hybrids in rust yield trials

Hybrid No.	Pedigree
Hi 662	(Ant2D X AA25) X (C123 X Mo17)
" 665	(CM111 X ") X (" X ")
" 669	(Ant2D X ") X (Va35 X C103D)
" 672	(CM111 X ") X (" X ")
" 676	(Ant2D X ") X (B37 X H84)
" 678	(CM111 X ") X (" X ")
" 681	(Ant2D X ") X (Hy2 X C103D)
" 688	(" X ") X (CM111 X CI21E)
" 691	(CM111 X ") X (Ant2D X B37)
" 695	(" X ") X (" X C103D)

A split-plot design with three replications and the hybrids as main plots and treatments as sub-plots (resistant vs. susceptible) were employed in two seasons. The hybrids segregated in a 1:1 ratio of

susceptible to resistant plants, since one of the four parents was heterozygous for Rp_1^d. When the plants were at the eight to nine leaf stage the segregating hybrids were thinned to leave one plant per hill. Each hybrid was represented by one row of resistant and one row of susceptible plants. Every plant susceptible to MMVI was also discarded from every plot before thinning. Each row was 4 meters long, with 20 cm between hills, and had 20 plants. Rows were separated by 76 cm.

An area of containing approximately 12 plants per plot (24 cm) was harvested at 7 weeks after mid-silking, and data collected on the following characteristics (averages were used for analysis of variance);

Rust rating : Average rust ratings of 10 plants based on 1 to 7 scale, 1 being highly generalized resistant.

Mid-silking : Average days from planting to silking for 10 individual plants.

Plant height : Average height in cm of 10 plants to tip of central tassel branch at five weeks after mid-silking.

Ear height : Average height in cm of the 10 plants from ground to node of uppermost ear.

Total fresh plant weight with ear : Fresh weight of plants in q/ha at harvest.

Fresh plant weight without ear : Total fresh plant weight minus ear weight.

Dry plant weight without ear (Stover weight) : Dry weight of the plant without ear after 7 days drying at 200°F inside oven, in q/ha.

Ear dimensions : Average length and mid-ear width in cm of 10 filled ears.

Kernel dimensions : Average diameter, width and depth in mm of 20 randomly selected kernels in the portion of center.

1000 kernel weight : Converted from the weight of 500 kernels at 15.0% moisture.

Grain yield : Total weight of grain in q/ha at 15.0% moisture.

Dry plant weight, kernel depth, kernel width, and kernel diameter were measured only from winter trial.

Statistical analyses were carried out for individual results in summer and in winter, and for data combined over two seasons according to individual characteristics.

The Waimanalo Farm is located 21 N in Oahu, Hawaii. The soil is a silty clay with pH 6.5. Fertilizer was applied at a rate of 600 lbs/acre of 16-16-16 before plowing and 300 lbs/acre urea as a side dressing after thinning. Sprinkler irrigation was used. Weather records at Waimanalo during summer and winter corn nurseries were as follows:

Season	Month	Rainfall in inches	Temperature in °F (Monthly Avg.) Max.	Min.
Summer	April	0.99	79	67
	May	1.76	80	67
	June	0.67	82	70
	July	2.71	83	71
	August	2.45	84	72
Winter	November	7.12	84	70
	December	7.29	78	66
	January	11.23	79	65
	February	3.60	79	67

RESULTS

Rust: Severe natural rust epiphytotics occurred in the corn nurseries at the Waimanalo Farm. Rust ratings of the 10 susceptible hybrids at mid-silking and three weeks after mid-silking in summer and winter are summarized in Table 2. In summer, an average rust rating of 4.6 based on the scale of 1 to 7 was produced at mid-silking, increasing to a rating of 5.2 at three weeks after mid-silking. In winter, the average rust ratings at mid-silking and three weeks after mid-silking were identical.

Analyses of variances of summer and winter are summarized in Table 3 and confirmed that the differences of rust ratings at mid-silking and three weeks after mid-silking (Treatment) were highly significant in summer, but not significant in winter. No significant differences were detected among 10 hybrids in both two seasons (Table 3).

Grain Yield: Grain yields of resistant and susceptible hybrids in quintals per hectare at 15.0% moisture, and percentages of reduction due to rust in summer, winter, and combined seasons are summarized in Table 4. In summer, average yields of the 10 paired resistant and susceptible hybrids were 118.0 and 86.4 q/ha, respectively, with an average of 26.6% yield reduction due to rust. In winter, yields were very poor compare to those in summer. The average yields of the resistant and susceptible hybrids were 36.9 and 20.9 q/ha, respectively, with an average of 42.8% yield reduction due to rust. A yield reduction of 30.8% due to rust occurred in the combined seasons, ranging from 16.7% for hybrid 695 to 48.9% for hybrid 672 (Table 4).

Analyses of variances of summer and winter are summarized in Table 5

Table 2. Rust ratings of ten hybrids at mid-silking and three weeks after mid-silking

	Rust (Scale 1-7)					
	Summer			Winter		
Hybrid	Mid-Silk	3 Weeks After M.S.	Change in rating	Mid-Silk	3 Weeks After M.S.	Change in rating
662	4.6	5.6	1.0	6.0	5.9	-0.1
665	4.9	5.1	0.2	5.9	6.2	0.3
669	4.6	5.3	0.7	5.9	6.1	0.2
672	4.6	5.5	0.9	6.1	5.9	-0.2
676	5.0	5.7	0.7	6.3	6.2	-0.1
678	4.9	5.8	0.9	6.3	6.0	-0.3
681	4.4	4.7	0.3	6.2	5.7	-0.5
688	4.5	5.1	0.6	5.4	6.0	0.6
691	4.5	4.9	0.4	6.1	6.0	-0.1
695	4.1	4.2	0.1	6.0	6.1	0.1
Avg.	4.6	5.2	0.6	6.0	6.0	0

Table 3. Analysis of variance of data in Table 2

		Mean square[+]	
Source	d.f.	Summer	Winter
Hybrid (H)	9	0.801	0.108
Rep (R)	2	0.748	0.766
Error (a)	18	0.431	0.204
Treatment (T)	1	4.817**	0.000
T X H	9	0.137	0.177
Error (b)	20	0.126	0.136

[+]Significance of mean square is indicated by *, $p < 0.05$ or by **, $p < 0.01$

Table 4. Grain yields of resistant and susceptible hybrids and reduction due to rust

	Grain yield (q/ha)				Percent reduction due to rust		
	Summer		Winter				Average
Hybrid	Res.	Sus.	Res.	Sus.	Summer	Winter	combined seasons
662	115.1	89.7	48.3	24.8	22.1	48.7	29.8
665	129.7	81.1	32.1	25.2	37.5	21.5	34.3
669	119.5	89.7	36.6	9.0	24.9	75.4	36.7
672	114.1	68.1	37.7	9.6	40.3	74.5	48.9
676	128.1	94.1	45.0	29.0	26.5	35.6	29.0
678	116.8	83.8	31.0	14.1	28.3	54.5	33.8
681	124.3	96.8	26.8	18.7	22.1	30.2	23.7
688	115.1	80.5	31.9	30.1	30.1	5.6	24.8
691	110.3	85.9	36.6	16.6	22.1	54.6	30.2
695	107.0	94.1	43.4	31.5	12.1	27.4	16.7
Avg.	118.0	86.4	36.9	20.9	26.6	42.8	30.8

BLSD 29.49 15.59

Table 5. Analysis of variance of data in Table 4

		Mean square		Combined seasons		
Source	d.f.	Summer	Winter	Source	d.f.	MS
Hybrid (H)	9	0.074	0.080*	Season (S)	1	55.135
Rep (R)	2	0.002	0.002	Rep (R)	2	0.004
Error (a)	18	0.046	0.033	Hybrid (H)	9	0.092**
				H x S	9	0.063
Treatment (T)	1	5.163**	1.326**	Error (b)	36	0.040
T X H	9	0.055	0.041			
Error (b)	20	0.040	0.041	Treatment (T)	1	5.861**
				T x S	1	0.628**
				T x H	9	0.047
				T x H x S	9	0.049
				Error (c)	40	0.041
				C.V. in %		16.60

and revealed highly significant differences between resistant and susceptible hybrids in both seasons. No significant differences were detected among the 10 hybrids in summer, but those in winter were significant. Analysis of variance of the combined seasons confirmed that highly significant differences occurred among rust treatments (Table 5). A highly significant treatment by season interactions (TxS) was detected. Differences among 10 hybrids in the combined season were also significant.

Total fresh plant weight: Total fresh plant weights with ear of resistant and susceptible hybrids in quintals per hectare at harvest, and percentages of reduction due to rust in summer, winter, and combined seasons are summarized in Table 6. In summer, average plant weights of the 10 paired resistant and susceptible hybrids were 493.9 and 366.7 q/ha, respectively, with an average of 25.8% weight reduction due to rust. In winter, plant weights were approximately half of those in summer. The average plant weights of the resistant and susceptible hybrids were 257.0 and 185.9 q/ha, with an average of 27.4% weight reduction due to rust. An average plant weight reduction of 26.5% due to rust occurred in the combined seasons, ranging from 18.0% for hybrid 695 to 37.2% for hybrid 672 (Table 6).

Analyses of variances of summer and winter are summarized in Table 7 and revealed highly significant differences among the 10 hybrids in both summer and winter, respectively. Analysis of variance of the combined seasons confirmed the highly significant differences among treatments (Table 7). A highly significant treatment by season interaction (TxS) was detected. Differences among 10 hybrids in the combined seasons were also highly significant.

Table 6. Total fresh plant weight with ear of resistant and susceptible hybrids and reduction due to rust

	Plant weight (q/ha)				Percent reduction due to rust		
	Summer		Winter				Average
Hybrid	Res.	Sus.	Res.	Sus.	Summer	Winter	combined seasons
662	458.4	328.6	269.2	160.5	28.3	40.4	32.7
665	468.1	350.8	213.0	173.0	25.1	18.8	23.1
669	489.2	363.8	249.7	172.4	25.6	31.0	27.5
672	506.5	293.0	231.4	169.7	42.2	26.7	37.2
676	518.9	373.0	257.8	199.5	28.1	22.6	26.3
678	482.7	315.1	217.8	147.6	34.7	32.2	34.0
681	523.2	440.0	244.9	188.6	15.9	23.0	18.2
688	538.9	415.7	323.8	231.4	22.9	28.5	25.0
691	448.6	372.4	300.5	202.2	17.0	32.7	23.3
695	504.9	414.6	261.6	214.1	17.9	18.2	18.0
Avg.	493.9	366.7	257.0	185.9	25.8	27.4	26.5
BLSD	90.5		47.6				

Table 7. Analysis of variance of data in Table 6

		Mean square		Combined seasons		
Source	d.f.	Summer	Winter	Source	d.f.	MS
Hybrid (H)	9	2.300*	1.666**	Season (S)	1	447.838
Rep (R)	2	1.681	4.722	Rep (R)	2	6.403
Error (a)	18	0.934	0.397	Hybrid (H)	9	3.153**
				H x S	9	0.813
Treatment (T)	1	83.261**	25.899**	Error (b)	36	0.666
T X H	9	0.879	0.265			
Error (b)	20	0.537	0.382	Treatment (T)	1	101.017**
				T x S	1	8.143**
				T x H	9	0.534
				T x H x S	9	0.610
				Error (c)	40	0.460
				C. V. in %		~~13.74~~ 11.41

Resistant hybrids retained their leaves and stalk in a relatively green state until the time of harvest, but susceptible hybrids lost most of their green tissues (Fig. 1). Disc samples also showed the differences (Fig. 2).

Fresh plant weight without ear: Fresh weights without ear of resistant and susceptible hybrids in quintals per hectare at harvest, and percentages of reduction due to rust in summer, winter, and combined seasons are summarized in Table 8. In summer, average fresh weights without ear of the 10 paired resistant and susceptible hybrids were 318.8 and 239.9 q/ha, respectively, with an average of 24.6% reduction due to rust. In winter, the average weight of the resistant and susceptible hybrids were 199.9 and 154.0 q/ha, with an average of 22.4% reduction due to rust. A weight reduction of 24.1% due to rust occurred in the combined seasons, ranging from 15.0% for hybrid 695 to 33.1% for hybrid 672 (Table 8).

Analyses of variances of summer and winter are summarized in Table 9 and revealed highly significant differences between treatments in both seasons. Significant differences were detected among 10 hybrids in both summer and winter. Analysis of variance of the combined seasons confirmed the highly significant treatment differences, and a highly significant treatment by and season interaction was detected. Differences among 10 hybrids in the combined seasons were also highly significant.

Dry plant weight without ear: Dry weights without ear of resistant and susceptible hybrids in quintals per hectare after drying 7 days at 200°F inside oven, and percentages of reduction due to rust in winter

Table 8. Fresh plant weight without ear of resistant and susceptible hybrids and reduction due to rust

	Fresh weight (q/ha)				Percent reduction due to rust		
	Summer		Winter				Average
Hybrid	Res.	Sus.	Res.	Sus.	Summer	Winter	combined seasons
662	289.7	211.9	199.8	126.7	26.9	36.6	22.2
665	295.7	231.4	164.5	138.9	21.7	15.6	30.8
669	296.2	233.5	196.2	155.9	21.2	20.5	30.8
672	334.6	193.5	180.2	151.0	42.2	16.2	33.1
676	324.9	234.6	185.2	155.9	27.8	15.8	21.0
678	298.4	197.3	167.9	125.4	33.9	25.3	24.3
681	338.4	300.5	204.7	160.9	11.2	21.4	19.6
688	361.1	290.3	266.5	185.0	19.6	30.6	20.6
691	289.2	239.5	238.7	171.7	17.2	28.1	23.5
695	351.9	266.5	194.8	168.1	24.3	13.7	15.0
Avg.	318.0	239.9	199.9	154.0	24.6	22.4	24.1

Table 9. Analysis of variance of data in Table 8

		Mean square				
		Fresh weight		Combined seasons		
Source	d.f.	Summer	Winter	Source	d.f.	MS
Hybrid (H)	9	1.678*	1.175*	Season (S)	1	106.993
Rep (R)	2	1.309	4.400	Rep (R)	2	5.709
Error (a)	18	0.568	0.345	Hybrid (H)	9	2.370**
				H x S	9	0.483
Treatment (T)	1	31.436**	10.821**	Error (b)	36	0.456
T X H	9	0.432	0.219			
Error (b)	20	0.404	0.268	Treatment (T)	1	39.572**
				T x S	1	2.685**
				T x H	9	0.218
				T x H x S	9	0.433
				Error (c)	40	0.336
				C. V. in %		11.24

are summarized in Table 10. Average dry weights of the 10 paired resistant and susceptible hybrids were 49.0 and 39.1 q/ha, respectively, with an average of 20.0% reduction due to rust. Analysis of variance revealed highly significant differences between treatments and among the 10 hybrids (Table 11).

1000 kernel weight: 1000 kernel weights of resistant and susceptible hybrids at 15.0% moisture, and percentages of reduction due to rust in summer, winter, and combined seasons are summarized in Table 12. In summer, average kernel weights of the 10 paired resistant and susceptible hybrids were 321.0 and 297.6 grams/1000 kernels, respectively, with an average of 7.0% reduction due to rust. In winter, kernel weight reduction was much greater than that in summer. The average 1000 kernel weights of the resistant and susceptible hybrids were 291.8 and 254.0 grams/1000 kernels, with an average of 12.8% reduction due to rust. Kernel weight reduction of 9.9% due to rust occurred in the combined seasons, ranging from 2.0% for hybrid 695 to 14.6% for hybrid 672 (Table 12).

Analyses of variances of summer and winter are summarized in Table 13 and revealed highly significant differences between treatments in both seasons. Differences among 10 hybrids in both summer and winter were highly significant. Analysis of variance of the combined seasons confirmed the highly significant treatment differences (Table 13). A highly significant hybrid X season interaction was detected.

Ear length: Ear lengths of resistant and susceptible hybrids, and percentages of reduction due to rust in summer, winter, and combined seasons are summarized in Table 14. In summer, average ear lengths of the 10 paired resistant and susceptible hybrids were 17.3 and 15.5 cm,

Table 10. Dry plant weight without ear of resistant and susceptible hybrids and reduction due to rust in winter

Hybrid	Dry weight (q/ha) Res.	Sus.	Percent reduction due to rust
662	44.9	34.1	24.1
665	42.7	30.8	27.9
669	48.6	37.3	23.3
672	48.6	43.2	11.1
676	47.6	41.1	13.7
678	43.2	33.5	22.5
681	55.1	40.5	26.5
688	57.8	42.7	26.1
691	54.1	43.8	19.0
695	47.0	44.3	5.7
Avg.	49.0	39.1	20.0

Table 11. Analysis of variance of data in Table 10

Source	d.f.	Mean square
Hybrid (H)	9	0.043*
Rep (R)	2	0.260
Error (a)	18	0.022
Treatment (T)	1	0.501**
T X H	9	0.008
Error (b)	20	0.017

Table 12. 1000 Kernel weight of resistant and susceptible hybrids and reduction due to rust

	1000 Kernel Weight (gr)				Percent reduction due to rust		
	Summer		Winter				Average
Hybrid	Res.	Sus.	Res.	Sus.	Summer	Winter	combined seasons
662	309.6	300.7	333.2	265.7	2.9	20.3	11.9
665	343.6	302.1	278.1	248.8	12.1	10.6	11.4
669	344.7	308.1	291.9	235.7	10.6	19.3	14.6
672	343.6	315.6	258.9	230.7	8.2	10.9	9.3
676	312.4	305.3	320.4	265.1	2.3	17.3	9.9
678	329.4	296.4	260.9	208.4	10.0	20.1	14.5
681	329.1	264.8	303.6	280.7	19.5	7.5	13.8
688	327.3	306.4	273.2	261.4	6.4	4.4	5.5
691	274.8	276.5	320.4	282.4	- 0.7	11.9	6.1
695	295.7	299.6	277.2	261.7	- 1.3	5.6	2.0
Avg.	321.0	297.6	291.8	254.0	7.0	12.8	9.9

Table 13. Analysis of variance of data in Table 12

		Mean square		Combined seasons		
Source	d.f.	Summer	Winter	Source	d.f.	MS
Hybrid (H)	9	1598.218*	3165.650**	Season (S)	1	39693.855
Rep (R)	12	289.001	167.370	Rep (R)	2	456.371
Error (a)	18	617.848	696.173	Hybrid (H)	9	840.827
				H x S	9	3921.041**
Treatment (T)	1	8259.093**	21369.143**	Error (b)	36	657.010
T X H	9	691.244	550.151			
Error (b)	20	301.228	1281.403	Treatment (T)	1	28099.063**
				T x S	1	1529.174
				T x H	9	541.905
				T x H x S	9	699.490
				Error (c)	40	791.315
				C.V. in %		9.66

Table 14. Ear length (filled) of resistant and susceptible hybrids and reduction due to rust

	Ear length (cm)				Percent reduction due to rust		
	Summer		Winter				Average
Hybrid	Res.	Sus.	Res.	Sus.	Summer	Winter	combined seasons
662	17.59	15.82	11.12	9.30	10.1	16.3	12.5
665	17.72	16.41	10.47	8.16	7.4	22.1	12.8
669	18.85	15.58	9.30	7.93	17.3	14.7	16.5
672	18.26	15.72	10.55	10.39	13.9	1.5	9.3
676	15.96	14.21	10.33	8.49	11.0	17.8	13.7
678	16.01	13.61	9.65	7.24	15.0	24.9	18.7
681	17.48	15.70	7.89	7.87	10.2	0.3	7.1
688	18.01	16.31	9.95	9.57	9.4	3.8	7.4
691	15.55	14.63	9.13	8.89	5.9	2.6	4.7
695	17.96	16.48	10.19	9.69	8.3	4.9	7.0
Avg.	17.34	15.45	9.86	8.75	10.8	10.9	11.0

BLSD 4.19 2.03

Table 15. Analysis of variance of data in Table 14

		Mean square		Combined seasons		
Source	d.f.	Summer	Winter	Source	d.f.	MS
Hybrid (H)	9	5.847**	4.085*	Season (S)	1	1507.334
Rep (R)	2	4.212	0.508	Rep (R)	2	4.720
Error (a)	18	0.589	1.440	Hybrid (H)	9	6.708**
				H x S	9	3.223**
Treatment (T)	1	53.582**	18.260**	Error (b)	36	1.014
T X H	9	0.685	1.333			
Error (b)	20	0.636	2.083	Treatment (T)	1	67.200**
				T x S	1	4.641
				T x H	9	1.153
				T x H x S	9	0.864
				Error (c)	40	1.360
				C.V. in %		9.68

respectively, with an average of 10.8% reduction due to rust. In winter, ear lengths were much shorter than those in summer. The average ear length of the resistant and susceptible hybrids were 9.9 and 8.8 cm, respectively, with an average of 10.9% reduction due to rust. Ear length reduction of 11.0% due to rust occurred in the combined seasons, ranging from 4.7% for hybrid 691 to 18.7% for hybrid 678 (Table 14).

Analyses of variances of ear lengths in summer and winter are summarized in Table 15 and revealed highly significant differences between treatments in both seasons. Highly significant differences were detected among 10 hybrids in summer, but those in winter were not significant. Analysis of variance of the combined seasons confirmed the significant treatment differences (Table 15). Highly significant hybrid X season interactions were also detected. Differences among 10 hybrids in the combined seasons were highly significant.

Ear diameter: Ear diameters of resistant and susceptible hybrids and percentages of reduction due to rust in summer, winter, and combined seasons are summarized in Table 16. In summer, average ear diameters of the 10 paired resistant and susceptible hybrids were 4.6 and 4.4 cm, respectively, with an average of 5.3% reduction due to rust. In winter, the average ear lengths of the resistant and susceptible hybrids were 4.0 and 3.4 cm, respectively, with an average of 14.0% reduction due to rust. Ear diameter reduction of 9.5% due to rust occurred in the combined seasons, ranging from 1.2% for hybrid 688 to 20.5% for hybrid 669 (Table 16).

Analyses of variances of summer and winter are summarized in Table 17 and revealed highly significant differences between treatments in both seasons. No significant differences were detected among 10 hybrids

Table 16. Ear diameter of resistant and susceptible hybrids and reduction due to rust

	Ear diameter (cm)				Percent reduction due to rust		
	Summer		Winter				Average
Hybrid	Res.	Sus.	Res.	Sus.	Summer	Winter	combined seasons
662	4.64	4.49	4.13	3.41	3.2	17.5	9.9
665	4.46	4.21	3.64	3.44	5.6	5.5	5.6
669	4.66	4.13	3.83	2.63	11.5	31.4	20.5
672	4.37	4.19	3.83	3.06	4.1	20.1	11.6
676	4.63	4.54	4.47	3.78	2.0	15.4	8.6
678	4.71	4.51	3.88	3.27	4.4	15.7	9.5
681	4.77	4.02	3.50	3.29	15.7	6.2	11.7
688	4.71	4.35	3.86	4.12	7.6	-6.7	1.2
691	4.44	4.57	4.54	3.66	-2.9	19.4	8.4
695	4.67	4.57	4.10	3.47	2.2	15.2	8.3
Avg.	4.61	4.36	3.98	3.41	5.3	14.0	9.5

Table 17. Analysis of variance of data in Table 16

		Mean square		Combined seasons		
Source	d.f.	Summer	Winter	Source	d.f.	MS
Hybrid (H)	9	0.088	0.573**	Season (S)	1	18.652
Rep (R)	2	0.157	0.002	Rep (R)	2	0.159
Error (a)	18	0.058	0.134	Hybrid (H)	9	0.471**
				H x S	9	0.191
Treatment (T)	1	0.935**	4.794**	Error (b)	36	0.096
T X H	9	0.092	0.254			
Error (b)	20	0.061	0.071	Treatment (T)	1	4.982**
				T x S	1	0.747**
				T x H	9	0.129
				T x H x S	9	0.217**
				Error (c)	40	0.066
				C.V. in %		6.29

in summer, but those in winter were highly significant. Analysis of variance of the combined seasons confirmed the significant treatment differences. Highly significant treatment X season and treatment X hybrid X season interactions were also detected. Differences among 10 hybrids in the combined seasons were highly significant (Table 17).

Kernel dimensions: Kernel diameters, widths, and depths of resistant and susceptible hybrids, and percentages of reduction due to rust in winter are summarized in Table 18. Average kernel diameters of the 10 paired resistant and susceptible hybrids were 8.5 and 8.0 mm, respectively, with an average of 5.9% reduction due to rust. Average kernel width of resistant hybrids was less than that of susceptible hybrids. The average kernel widths of the resistant and susceptible hybrids were 4.8 and 5.2 mm, respectively, with an average of 9.0% difference. Average kernel depths of the resistant and susceptible hybrids were 9.8 and 8.7 mm, respectively, with an average of 10.5% reduction due to rust.

Analyses of variances of the kernel dimensions are summarized in Table 19 and revealed highly significant differences between resistant and susceptible hybrids for kernel diameter and kernel depth. By contrast, a significant kernel width increase related to rust occurred in susceptible hybrids, perhaps reflecting a decrease in number of kernels per row and number of ears per plot (unstudied). No significant differences were detected among 10 hybrids for all kernel dimensions.

Plant height: Plant heights of resistant and susceptible hybrids to tip of central tassel branch, and percentages of reduction due to rust in summer, winter, and combined seasons are summarized in Table 20. In summer, average heights of the 10 paired resistant and susceptible

Table 18. Kernel diameter, kernel width, and kernel depth of resistant and susceptible hybrids and reduction due to rust in winter

Hybrid	Diameter (mm) Res.	Diameter (mm) Sus.	Percent reduction	Width (mm) Res.	Width (mm) Sus.	Percent reduction	Depth (mm) Res.	Depth (mm) Sus.	Percent reduction
662	9.3	8.5	8.6	5.0	4.8	4.0	10.5	9.4	10.5
665	8.8	8.7	1.1	4.4	4.8	9.1	9.6	9.1	5.2
669	8.6	7.2	16.3	4.9	5.9	-20.4	9.3	7.7	17.2
672	7.8	8.3	-6.4	4.8	5.6	-16.7	9.6	7.6	20.8
676	8.7	7.9	9.2	4.9	4.8	2.0	10.6	9.7	8.5
678	8.0	7.5	6.3	4.4	5.3	-18.2	9.4	9.0	4.3
681	7.6	8.2	-7.9	4.9	5.3	- 8.2	10.5	8.8	16.2
688	8.9	7.7	13.5	5.1	4.9	3.9	8.7	9.5	-9.2
691	9.4	7.9	16.0	4.8	6.2	-29.2	10.4	7.8	25.0
695	8.1	7.9	2.5	4.9	4.8	2.0	9.4	8.8	
Avg.	8.5	8.0	5.9	4.8	5.2	- 9.0	9.8	8.7	10.5

Table 19. Analysis of variance of data in Table 18

Source	d.f.	Kernel Diameter	Kernel Width	Kernel Depth
Hybrid (H)	9	0.919	0.444	1.706
Rep (R)	2	0.269	0.006	0.007
Error (a)	18	0.380	0.568	1.491
Treatment (T)	1	4.817**	2.646*	16.960**
T X H	9	0.850	0.535	1.385
Error (b)	20	0.490	0.376	1.066

Table 20. Plant height of resistant and susceptible hybrids and reduction due to rust

	Plant height (cm)				Percent reduction due to rust		
	Summer		Winter				Average
Hybrid	Res.	Sus.	Res.	Sus.	Summer	Winter	combined seasons
662	262.7	267.2	187.9	171.5	-1.7	8.7	2.6
665	275.3	283.3	183.1	171.0	-2.9	6.6	0.9
669	286.0	279.3	180.3	171.2	2.3	5.1	3.4
672	299.7	275.0	189.4	173.9	8.2	8.2	8.2
676	279.3	275.7	182.1	177.3	1.3	2.6	1.8
678	286.0	264.7	177.5	161.1	7.5	9.2	8.1
681	269.7	280.7	191.7	173.8	-4.1	9.3	1.5
688	289.3	288.3	191.3	183.3	0.4	4.2	1.9
691	268.3	273.6	206.8	187.3	-2.7	9.4	3.0
695	296.9	287.7	193.1	190.3	3.1	1.5	2.5
Avg.	281.3	277.5	188.3	176.1	1.1	6.5	3.4

Table 21. Analysis of variance of data in Table 20

		Mean square		Combined seasons		
Source	d.f.	Summer	Winter	Source	d.f.	MS
Hybrid (H)	9	431.077*	390.529*	Season (S)	1	283629.633
Rep (R)	2	263.517	408.686	Rep (R)	2	672.203
Error (a)	18	131.794	137.969	Hybrid (H)	9	485.790**
				H x S	9	335.817*
Treatment (T)	1	213.571	2247.264**	Error (b)	36	134.881
T X H	9	216.332	50.380			
Error (b)	20	109.91	48.604	Treatment (T)	1	1923.201**
				T x S	1	537.633*
				T x H	9	118.552
				T x H x S	9	148.160
				Error (c)	40	83.191
				C.V. in %		~~3.86~~ 3.95

hybrids were almost identical, with an average of 1.1% height reduction due to rust. Plant heights of the 4 out of the 10 hybrids were increased slightly due to rust. In winter, plant heights were affected greatly by rust. The average plant heights of the resistant and susceptible hybrids were 188.3 and 176.1 cm, respectively, with an average of 6.5% reduction due to rust. A plant height reduction of 3.4% due to rust occurred in the combined seasons, ranging from 0.9% for hybrid 669 to 8.2% for hybrid 672 (Table 20).

Analyses of variances of summer and winter are summarized in Table 21 and revealed no significant difference among treatments in summer. However, the difference was highly significant in winter. Significant differences were detected among 10 hybrids in both seasons. Analysis of variance of the combined seasons confirmed the highly significant treatment differences (Table 27). A significant treatment X season (TxS) interaction was detected. Differences among 10 hybrids in the combined seasons were also highly significant. A significant hybrid X season interaction was also detected.

Ear height: Ear heights of resistant and susceptible hybrids to node of first ear, and percentages of reduction due to rust in summer, winter, and combined seasons are summarized in Table 22. In summer, average ear heights of the 10 paired resistant and susceptible hybrids were almost equal, and were 116.6 and 113.9 cm, respectively. In winter, the average ear heights of the resistant and susceptible hybrids were 75.6 and 69.4 cm, respectively, with an average of 8.2% reduction due to rust. Ear height reduction of 4.6% due to rust occurred in the combined seasons, ranging from -0.9% hybrid 688 to 8.2% for hybrid 672.

Table 22. Ear height of resistant and susceptible hybrids and reduction due to rust

	Ear height (cm)				Percent reduction due to rust		
	Summer		Winter				Average
Hybrid	Res.	Sus.	Res.	Sus.	Summer	Winter	combined seasons
662	106.1	108.0	72.0	64.0	1.8	11.1	3.4
665	106.4	105.7	69.8	62.0	0.7	11.1	4.8
669	124.3	114.9	77.7	72.1	7.6	7.1	7.4
672	120.0	107.7	74.2	70.6	10.3	4.9	8.2
676	108.5	108.5	68.5	69.7	0.0	-1.7	-0.6
678	112.0	103.0	73.7	59.2	8.0	19.6	12.6
681	126.5	127.5	76.3	67.6	-0.8	11.4	3.8
688	120.9	127.5	86.3	81.7	-5.5	5.4	-0.9
691	113.4	114.7	81.0	76.3	-1.1	5.8	1.8
695	127.4	121.8	76.7	71.0	4.4	7.4	5.5
Avg.	116.6	113.9	75.6	69.4	2.2	8.2	4.6

Table 23. Analysis of variance of data in Table 22

		Mean square		Combined seasons		
Source	d.f.	Summer	Winter	Source	d.f.	MS
Hybrid (H)	9	391.250**	193.535**	Season (S)	1	54754.224
Rep (R)	2	119.150	14.873	Rep (R)	2	134.023
Error (a)	18	100.411	41.077	Hybrid (H)	9	479.361**
				H x S	9	105.425
Treatment (T)	1	103.491	575.980**	Error (b)	36	70.744
T X H	9	55.694	24.283			
Error (b)	20	31.279	47.247	Treatment (T)	1	583.884**
				T x S	1	95.587
				T x H	9	46.726
				T x H x S	9	33.250
				Error (c)	40	39.263
				C.V. in %		6.68

Analyses of variances of summer and winter are summarized in Table 23 and revealed no significant differences for ear height between resistant and susceptible hybrids in summer, but highly significant differences were detected in winter. Highly significant differences were detected among 10 hybrids in both seasons. Analysis of variance of the combined seasons confirmed that highly significant differences occurred in treatments and among 10 hybrids (Table 23).

Mid-silking: Days to mid-silk of resistant and susceptible hybrids, and percentages of reduction due to rust in summer, winter, and combined seasons are summarized in Table 24. In summer, average days to mid-silk of the resistant and susceptible hybrids were 63.6 and 64.0 days, respectively, with an average of approximately half day delay due to rust. In winter, the average days to mid-silk of the resistant and susceptible hybrids were 62.2 and 64.7 days, respectively, with an average of approximately two and half days delay due to rust.

Analyses of variance of summer and winter, summarized in Table 25 revealed significant differences between resistant and susceptible hybrids in summer, but the differences were highly significant in winter. Highly significant differences were detected among the 10 hybrids in both two seasons. Analysis of variance of the combined seasons confirmed the highly significant treatment differences (Table 25). A highly significant treatment X season interaction and significant treatment X hybrid interactions were detected. Differences among 10 hybrids in the combined seasons were highly significant. A significant hybrid X season interaction was also detected.

Table 24. Days to mid-silk of resistant and susceptible hybrids and delay due to rust

	Days to mid-silk				Days delay due to rust		
	Summer		Winter				Average
Hybrid	Res.	Sus.	Res.	Sus.	Summer	Winter	combined seasons
662	62.0	62.3	60.7	61.3	0.3	0.6	0.5
665	62.0	62.0	59.0	61.7	0.0	2.7	1.3
669	63.7	64.7	63.3	68.3	1.0	5.0	3.0
672	64.0	64.0	62.0	63.7	0.0	1.7	0.7
676	64.3	64.7	61.7	64.0	0.4	2.3	1.3
678	63.7	64.7	63.0	64.7	1.0	1.7	1.4
681	64.0	64.0	61.0	63.0	0.0	2.0	1.0
688	64.7	65.0	65.7	69.0	0.3	3.3	1.9
691	64.0	64.3	62.7	66.3	0.3	3.6	2.0
695	64.0	64.3	63.3	64.7	0.3	1.4	0.8
Avg.	63.6	64.0	62.2	64.7	0.4	2.4	1.4

Table 25. Analysis of variance of data in Table 24

		Mean square		Combined seasons		
Source	d.f.	Summer	Winter	Source	d.f.	MS
Hybrid (H)	9	5.363**	27.261**	Season (S)	1	3.675
Rep (R)	2	0.650	4.050	Rep (R)	2	4.700
Error (a)	18	0.335	5.439	Hybrid (H)	9	25.968**
				H x S	9	6.657*
Treatment (T)	1	1.667*	88.817**	Error (b)	36	2.887
T X H	9	0.259	2.446			
Error (b)	20	0.300	1.133	Treatment (T)	1	57.408**
				T x S	1	33.075**
				T x H	9	1.6491*
				T x H x S	9	1.057
				Error (c)	40	0.717
				C.V. in %		1.33

DISCUSSION

Grain yield reduction due to rust in these experiments under severe rust epiphytotics were much higher, ranging from 16.7 to 48.9%, than those previously reported. Hooker (1962) reported a maximum yield reduction due to rust of 6.3% in a susceptible version of hybrid B14 X Oh41. Russell (1965) reported 19.8% average yield reduction due to rust in three susceptible hybrids involving B14A. Hybrid 672 showed very high yield reductions in both seasons. Highly susceptible parents C103D and Va35 (Table 26) might cause the yield reductions, since the barrenness in crosses involving C103D has been reported (Eberhart and Russell 1969).

Fresh and dry plant weight reductions, averaging 26.5%, under severe rust epiphytotics would clearly be of economic significance in silage production in Hawaii. The moisture in tissues of susceptible hybrids decreases earlier than in resistant counterparts. A similar observation was made by Russell (1965), who reported that rust resistant hybrids had 3.6% more moisture in grain than their susceptible counterparts, indicative of slower maturation.

Environmental conditions in summer and in winter were quite different, but it was clear that the rust affected grain yields and other agricultural characteristics similarly in the two seasons. Error mean squares for grain yield in summer (.0400) and in winter (.0408) were almost equal, despite great discrepancy in the average reductions (27 vs 43%), indicating the feasibility of combined analyses of variance. Year-round corn culture and sprinkler irrigation enhance rust epiphytotics in Hawaii. Rust started to infect the corn leaves earlier in winter than

in summer, favored by low temperatures and high humidity, but later rust ratings of different hybrids were very similar in the two seasons.

Highly significant 1000 kernel weight reductions due to rust would be important yield component reduction. The highest grain yield hybrid 695 had the least kernel weight reductions and the lowest grain yield hybrid 672 had the greatest kernel weight reductions in the combined seasons. Significant kernel weight reductions due to Helminthosporium turcicum and Helminthosporium maydis in maize have been reported by Hooker and Kim (1973) and by Lim et al. (1974) respectively. Hooker and Kim observed significant kernel weight differences between H. turcicum resistant hybrids containing the Ht gene and their susceptible counterparts. Lim et al. observed significant grain yield and 1000 kernel weight differences between hybrids with Texas male-sterile cytoplasm (cms-T) and their normal-cytoplasm counterparts. The amount of grain yield and kernel weight of cms-T hybrids were significantly correlated with those of their normal-cytoplasm counterparts. In the case of rust highly significant reductions of ear length and ear diameter due to rust were not directly related to grain yield reductions.

Highly significant treatment X season and treatment X hybrid X season interactions occurred in ear diameter, indicating a large amount of treatment and hybrid differences over two seasons. These interactions may result in no direct correlation between grain yield and ear diameter. The importance of row number and kernels per row was recognized in this study, but most ears in both resistant and susceptible hybrids had identical row numbers (unsummarized data). Although kernels per row were not measured, they might be highly correlated with ear length and grain yield reduction.

Plant height and ear height reductions due to rust are not considered as important factors in double-cross hybrids. However, in winter at the Waimanalo Farm, when corn leaves were heavily infected with P. sorghi at the young vegetative stage, the plant height of susceptible inbreds and hybrids seemed to be reduced greatly.

Delay of mid-silking due to rust in winter was somewhat greater than in summer. It was also observed that highly susceptible sweet corn inbreds AA8 and 442 were delayed about four days in silking over their resistant counterparts: $AA8Rp_1{}^dBC_6$ and $442Rp_1{}^dBC_5$ in winter, 1973.

Highly significant treatment X season interactions were detected for grain yield, total fresh plant weight with and without ear, ear diameter, and mid-silking. Using the T x H x S interaction as an error, there were also highly significant treatment X season interactions for grain yield, total fresh plant weight with and without ear (only at the 5% level), and mid-silking, indicating that the rust generally affected these traits more severely in the winter when growth was otherwise poor. There were no significant treatment X season interactions detected for kernel weight, ear length, and ear height.

The difficulties in measuring treatment effects with disease resistant Ht genes to H. turcicum in maize has been illustrated (Ullstrup 1970). There are several reports in which genotype X environment interactions were examined in corn by Eberhart and Russell (1969), cotton by Miller et al. (1959), tobacco by Jones et al. (1960), wheat, barley, and oat by Liang et al. (1966), and sorghum by Liang and Walter (1966), but a limited number of references on treatment effects with isogenic resistant lines is available. Russell (1965) found significant treatment X environment interactions in maize yield to P. sorghi.

The results obtained in this study give an indication of the significant potential damage on corn yield that may be caused by P. sorghi in tropics and subtropics as well as temperate regions. Borlaug (1965) reported that double-cross hybrids in Mexico and Central America seemed to be attacked much more severely by rusts than were the open-pollinated and synthetic varieties. Hooker (1969) cautioned that the increase in the commercial use of single-cross hybrids in the U. S. Corn Belt may reduce genetic diversity, and is potentially dangerous as it may increase susceptibility to P. sorghi.

II. SOURCES OF RUST RESISTANCE IN MAIZE

ABSTRACT

Severe natural epiphytotics of common corn rust, Puccinia sorghi Schw., were obtained by continuous year-round corn plantings in Hawaii. P. sorghi races present in these nurseries were shown to pathogenic on all monogenic sources except six Rp_1 sources a, d, d (2), f, g, and k.

Rust observations were made by cooperators on a series of our lines in Hawaii, Mexico, Thailand, and Illinois. Two Puccinia polysora resistant lines were highly susceptible to P. sorghi in Hawaii. Seven of our 15 generalized resistant entries were highly resistant to P. sorghi in Mexico. Most of the P. sorghi resistant entries were susceptible to P. polysora in Thailand. The best generalized resistance source CM105 was highly resistant to P. sorghi in Hawaii, Mexico, and Illinois. It was also resistant to P. polysora in Thailand.

Forty inbred lines were scored on an individual-plant basis in four different seasons for generalized resistance to P. sorghi, and 41 tropical races of maize were evaluated. Only two inbreds showed outstanding generalized resistance in Hawaii. They were Oh545 (Ohio) and CM105 (India). Most US dent inbreds were scored as highly susceptible in all tests. Forty-two inbred lines were scored weekly for their generalized resistance from three weeks before mid-silking to four weeks after mid-silking. Highly resistant inbreds showed a tendency toward resistance at one week before mid-silking, and continued to exhibit this ontogenetic change until two weeks following mid-silking. However, susceptible inbreds developed lesions continuously throughout the experimental period.

Maize races exhibited differing degrees of resistance, some showing specific or fleck-type resistance. Races with high generalized resistance included ancient Polomero Toluqueno, along with more racent races Amagaceno, Clavo, Comiteco, Imbricado, and Sabanero.

A genetic study was made to determine gene action conditioning Puccinia sorghi resistance in sweet corn inbred 677a. The 677a resistance was interpreted as monogenic and recessive. Phenotypic expression of this resistance was unusual, showing reddish-purple flecking, often becoming necrotic. In general the resistance was developed fully at flowering time. Progeny tests involving the dominant resistance gene, Rp_1^d, suggested that the recessive resistance gene in inbred 677a is an allele of one of the five Rp loci located on chromosome 10 in maize.

MATERIALS AND METHODS

1. Monogenic Rust Resistance

Thirty monogenic sources of rust resistance obtained from Dr. A. L. Hooker of the University of Illinois were used in this study. In general, all plots were single rows containing approximately 15 to 20 plants spaced 8 inches apart, with rows spaced 30 inches apart. 600 lb/acre of 16-16-16 fertilizer was applied before planting, and 300 lb/acre of 16-4-4 fertilizer about one month after emergence. Sprinkler irrigation systems were used. All plants were subjected to natural rust epiphytotics. Average rust ratings were taken at mid-silking date from each plant in the plot.

2. International Rust Observations

Thirty-four generalized and specific rust resistant seed stocks were tested in Hawaii, Mexico, Thailand, and Illinois in 1972. Cooperators were Dr. C. D. Leon, CIMMYT, Mexico, Dr. C. L. Moore, Rockefellow Foundation, Thailand, and Dr. A. L. Hooker, University of Illinois, Illinois.

Hawaii: Twenty-nine *P. sorghi* and 3 *P. polysora* entries were planted at the Waimanalo Farm on January 4th and September 27th in 1972. All plants were subjected to natural rust epiphytotics prevalent on the farm. Individual plants were scored on a 1 to 5 scale for generalized resistance, and ratios of segregating entries for specific resistance were calculated.

Mexico: Thirty-four rust entries were tested at Chapingo and were inoculated artificially with *P. sorghi* uredio-spores. Average rust data were based on the scale 1 to 5 at approximately four weeks after mid-silking.

Thailand: Twenty-six rust entries were planted on August 9, 1972 at Bangkok and inoculated at flowering time with P. polysora collected from a previous crop. Rust ratings were taken on October 31st from individual plots based on the scale 1 to 5.

Illinois: Twenty-two rust entries were planted at the University South Farm, Champaign-Urbana and inoculated at 7 to 8 leaf stage with P. sorghi spores collected from a previous crop. All individual plants were scored at two weeks before mid-silking and at approximately five weeks after mid-silking. At two weeks before mid-silking, the author took data based on the scale 1 to 5, 1 being highly resistant and 5 being highly susceptible for generalized resistance and took data based on the ratio of individual plants in entries segregating for specific resistance. At five weeks after mid-silking, Dr. Hooker took the data based on the percentage of leaf area infected with rust. A highly susceptible check hybrid was planted as hill plot at the end of each plot.

3. Sources of Generalized Rust Resistance

Forty widely used inbred lines of maize were selected for this study and grown in four different seasons at the Waimanalo Farm since 1972. Individual plots were single rows containing 20 plants. All plants were subjected to natural rust epiphytotics. Rust ratings were taken about three weeks after mid-silking from 20 individual plants in each plot.

The rating scales for leaf rusts of cereals proposed by Peterson et al. (1946) and James (1972) were modified for scoring the degree of rust ratings based on the scale 1 to 7, comparing with resistant and susceptible check plants (Fig. 3).

4. Ontogenetic Changes of Generalized Resistance

Forty-two inbred lines planted March 8, 1973, were scored weekly for their generalized resistance based on a severity scale 1 to 7 from three weeks before mid-silking to four weeks after mid-silking.

5. Maize Race Screening Test

Fifty tropical maize races obtained from CIMMYT, Mexico, and CIAT, Colombia, were screened for their rust reactions under natural rust epiphytotics in two different seasons. Rust ratings were made in May, 1972 and in August 1973. Individual plots were single rows containing approximately 15 plants.

6. 677a Resistance

Sweet corn inbred line 677a was developed by Dr. A. M. Rhodes from a 3-way cross (Bolivia 1035 X 44b) X 442a and released corn breeders by the University of Illinois in 1972 (Gonsales et al. 1972). Under rust infection in Hawaii, 677a showed reddish-purple fleckings, with limited P. sorghi spore production, on corn leaves. The inbred 677 and the three following tester lines were used in this study:

AA8 = Sweet corn inbred (Hawaii Agr. Exp. Sta.)

442 = Sweet corn inbred (Illinois Agr. Exp. Sta.)

Rp_1^d = Single dominant rust resistant source from international rust materials (Dr. A. L. Hooker).

677a was crossed with each tester line, and F_1 crosses were advanced to the F_2 or BC generations by pollinating approximately 10 F_1 plants. All consisted of 4 parents, $3F_1$'s, $3F_2$'s, and 3 backcrosses were grown at the Waimanalo Farm during 1972 and 1973. All plants were subjected to a natural rust inoculum and evaluated for disease reaction (resistant vs susceptible) at flowering time. The 4 parents were used

as check lines in disease evaluations.

Chi-square tests were employed to estimate goodness of fit of the segregating F_2 and backcross populations.

RESULTS AND DISCUSSION

1. Monogenic Rust Resistant Sources

Thirty international rust lines carrying monogenic resistance to P. sorghi were tested in three different seasons at the Waimanalo Farm Oahu, and one season at the Lalamilo Farm, Hawaii (Table 26). Planted at the Waimanalo Farm in 1971, eleven Rp_1 sources a, b, c (2), c-k, d, d (2), f, g, j, k, and m, and an unidentified allele or locus in line 192 conferred complete, fleck-type resistance. Two additional Rp_1 alleles, c, and i, conferred partial or moderate resistance. All other sources were susceptible (Table 26). Similar results were obtained in 1972, except that Rp_1^m showed partial resistance. In 1973, four Rp_1 sources (b, c (2), c-k, and j) appeared no longer to confer complete resistance. On the island of Hawaii, at the Lalamilo Farm in Waimea in 1971, only 6 of the 10 Rp_1 sources a, d, d (2), f, g, and j conferred complete resistance (Table 26).

Rust was first observed at the Waimanalo Farm in 1967 and these data (Table 26) suggest an increasing severity or changing racial composition of this rust.

2. International Rust Observations in 3 Countries

Hawaii: The results of P. sorghi epiphytotics on 24 generalized and 12 specific resistance entries are summarized in Table 27. CM105 and crosses with CM105 evidenced good generalized resistance to P. sorghi. However, AA8, CM104, and their propenies were highly susceptible. Four tested Rp_1 alleles, b, d, d (2), and g in their progeny tests were completely resistant to rust and were controlled by a single dominant gene. These Rp data are supported by the results from the International Rust Program (Table 26).

Table 26. Average rust ratings at mid-silking of 30 international monogene rust maize lines planted on four different planting dates

Line	Planting dates 1*	2	3	4	Line	Planting dates 1	2	3
1. Check (sus)	+**	++	+++	++	16. $Rp_3{}^a$	+	+	
2. $Rp_1{}^d$	-	-	-	-	17. $Rp_3{}^b$	++	+	
3. $Rp_1{}^b$	-	-	+	++	18. $Rp_3{}^e$	++	+	
4. $Rp_1{}^f$	-	-	-	-	19. $Rp_4{}^a$	++	+	
5. $Rp_1{}^c$	+	+	++		20. $Rp_4{}^b$	++	+	
6. $Rp_1{}^k$	-	-	-		21. Rp_5	++	+	
7. $Rp_1{}^{c-k}$	-	-	++	++	22. $Rp_1{}^h$	++	++	
8. $Rp_1{}^a$	-	-	-	-	23. $Rp_1{}^l$	+	+	+
9. rp_2	++	+	++		24. $Rp_3{}^d$	+	++	
10. $Rp_3{}^c$	+	+			25. $Rp_3{}^f$	++	+	
11. $Rp_1{}^{d(2)}$	-	-	-	-	26. AAN	+	+	++
12. $Rp_1{}^g$	-	-	-	-	27. 82	++	+	
13. $Rp_1{}^{c(2)}$	-	-	+	++	28. 39-25	+	+	++
14. $Rp_1{}^j$	-	-	++	-	29. 192	++	+	++
15. $Rp_1{}^m$	-	+	++	+	30. 182	-	-	++

Planting date	Place
*1=April 22, 1971	Waimanalo
2=Aug. 25, 1972	Waimanalo
3=Feb. 6, 1973	Waimanalo
4=Sept. 27, 1971	Lalamilo

**Minus (-) means fleck reaction, and +, ++, and +++ mean relative susceptibility from slight to severe.

Two Puccinia polysora resistant lines and one susceptible line were also grown in September, 1972. Both P. polysora resistant lines were highly susceptible to P. sorghi, recorded as 5 on a scale of 1 to 5. The susceptible check line showed some level of generalized resistance to P. sorghi. These results support the mycological evidence that only P. sorghi is present at the Waimanalo Farm.

Mexico: Average rust ratings of the 43 entries to P. sorghi at Chapingo, Mexico, are summarized in Table 27. Seven of the 15 generalized resistant entries were rated as 1 to 2 based on the scale of 1 to 5, 1 being highly resistant, but 8 entries rated as 3 to 5. Backcross (AA8 X CM104) CM104 rated as 1 and was identified as the best resistant entry to P. sorghi in Mexico. By contrast, this backcross was highly susceptible in Hawaii. Inbreds CM104 and CM105 rated as 2. The variability of CM105 for generalized resistance was reported. Crosses with CM105 were not resistant. AA8 was highly susceptible both in Mexico and in Hawaii. It is not easy to interpret clearly the data obtained in the monogenic resistance because several entries were heterogeneous. However these data clearly indicated that three Rp_1 alleles, b, d, and d (2) were completely resistant to P. sorghi in Mexico. Rp_1^g was susceptible in Mexico but confers resistance in Hawaii.

Thailand: The inoculation was made very late, at flowering time. It was reported that some natural infection occurred late in the season and differences could be detected between entries. The results of rust ratings shown in Table 27 indicated that CM104 and AA8 X CM104 possessed high resistance to rust. Puccinia polysora might be present only in Thailand, with a remote possibility that P. sorghi is also present, because the highly resistant entries shown in Thailand are very

susceptible to P. sorghi in Hawaii. CM105 was highly resistant but crosses with CM105 were susceptible. It was hard to interpret clearly the data obtained in the monogenic resistance Rp_1 entries. However, Rp_1 alleles b, d, d (2), and g conferred no resistance to P. polysora in Thailand. Their F_1's and backcrosses were all susceptible.

Illinois: Two weeks before mid-silking, the average rust ratings of the 16 entries were taken for generalized resistqnce, and ratios in populations segregating specific resistance (Table 27). CM105 and crosses with CM105 such as CM105 X CM104 showed generalized resistance. Only one AA8 plant was available, and seemed to have escaped rust infection. Two backcrosses, (CM105 X AA8) AA8 and (CM105 X CM104) CM104 were susceptible. At this time, it was too early to take rust ratings for generalized rust resistance. The results of 7 entries in the monogenic resistance are summarized in Table 2, and clearly indicate that Rp_1 alleles d and d (2) were completely resistant to rust in Illinois. These results were supported by Hooker's several reports (Hooker 1967, Hooker 1969).

Five weeks after mid-silking, the average percent of leaf area covered by rust per plot was scored (Table 27). CM105 had high generalized resistance. AA8 showed high resistance, as did such crosses as AA8 X CM104, contrasting sharply with their high susceptibility in Hawaii. By comparison with the results obtained 2 weeks before mid-silking, only one AA8 plant seemed to have escaped rust infection. Hence the time of rust ratings seemed to be too late for relatively early materials such as AA8 and AA8 X CM104.

Table 27. Average rust ratings on generalized and specific resistant entries tested in Hawaii, Mexico, Thailand, and Illinois in 1972 (1-5 scale); P. polysora = Thailand

Entry	Hawaii +4 weeks	Mexico +4 weeks	Thailand +4 weeks	Illinois -2 weeks	Illinois +5 weeks* %
Generalized res.					
AAh	4.8	4+	Tr+	2.0	Tr+
CM104	4.9	2	1.2	2.5	5.0
CM105	2.0	2	1.1	2.3	1.6
AA8 X CM104	4.5	2	1.2	3.0	1.0
CM105 X AA8	1.8	4	2.0	1.8	1.9
CM105 X CM104	2.9	3	2.0	1.5	2.6
(AA8 X CM104) x	4.6	2	2.0	3.0	17.5
(CM105 X AA8 x	2.9	3	2.3	2.5	9.5
(CM105 X CM104 x	4.2	4	1.2	1.8	5.7
(AA8 X CM104)AA8	4.9	4	1.5	2.0	15.4
(AA8 X CM105)AA8	4.5	2	1.5	3.3	16.3
(AA8 X CM104)CM104	4.8	1	1.1	2.5	2.8
(CM105 X CM104)CM104	4.1	2	1.2	3.3	4.1
(CM105 X AA8)CM105	2.5	4	2.0	2.0	3.7
(CM105 X CM104)CM105	3.3	3	2.0	1.8	6.1
HS Syn2	4.0	2			
KCo # 1 X HS RRCompl	4.0	4		3.0	
Specific res.	R : S**			R : S	R : S
KCo # 2 X AA8sh2 Rp_1^d	25 : 0	4			
Rp_1^d X AA8	28 : 0	2	2.5	2 : 0	2 : 0
Rp_1^d X CM104	40 : 0	1	2.5	9 : 0	9 : 0
Rp_1^d X CM105	51 : 0	2	2.5	11 : 0	11 : 0
(Rp_1^d X AA8sh2)AA8sh2	16 : 18	5	3.5		
(Rp_1^d X HS syn2)Hs syn2	38 : 45	2	3.5	10 : 7	10 : 7
(Rp_1^d X CM104)CM104	62 : 41	5	2.5	7 : 7	7 : 7
($Rp_1^{d(2)}$ X AA8)AA8	20 : 24	2	3.5	6 : 8	6 : 6
($Rp_1^{d(2)}$ X HSSyn2)HSSyn	38 ; 45	2	3.5	3 : 4	3 : 4
(Rp_1^g X AA8)AA8	21 : 23	4	5.0		
(Rp_1^g X 442a)442a	14 : 19	4	4.5		
(Rp_1^b X AA8sh2)AA8sh2	11 : 14	2	3.5		
Ant2 Gp.2 X B37		1			
Rp_1^b		2			
Rp_1^d		2			
$Rp_1^{d(2)}$		2			
Rp_1^g		4			
P. polysora resistant materials					
Susceptible check	2				
Caqueta Comp. (resistant)	5				
Zap. Chico (Juchitan 70) (resistant)	5				

*Weeks in relations to mid-silking.
**Number of plants in resistant and susceptible.
+Ratings supplied by cooperators.

3. Generalized Rust Resistant Sources

Forty widely used U. S. and foreign inbred lines of corn were tested in a search for better generalized resistance sources to P. sorghi in four different seasons at the Waimanalo Farm between 1972 and 1974. Rust ratings were made approximately three weeks after mid-silking based on individual plants. The forty inbred lines varied greatly in their level of generalized rust resistance (Table 28). On a severity scale of 1 to 7 (1 being highly resistant), 21 of the 40 lines rated above 5 and these were identified as highly susceptible sources to P. sorghi. Five lines rated under 3, or highly resistant; these lines were Oh545, CM105, CM111, N6G, and Antigua 2D (Table 28). Two lines, Oh545 (Ohio, dent) and CM105 (India, flint), were identified with outstanding generalized resistance in all four seasons. The rust ratings of the different inbred lines in different seasons were quite consistent, indicating that environmental effects were relatively small through the seasons sampled.

The most susceptible inbred lines, B37 (Fig. 4) and PH9 (Fig. 5)has approximately 90% leaf area covered by rust. Not only B37 but B14 and other popular inbred lines in the U. S. Corn Belt and other regions were severely damaged by rust. It is noteworthy that inbreds B73 (from B37) and B68 (from B14) were more resistant than their parent inbreds. The high susceptibility of B14 had been reported previously by Hooker (1962) and by Russell (1965). However, other popular inbred lines, such as Oh43, Va26, and Oh545 (Fig. 5) were much less susceptible. Thus, considerable choice exists in parent selection for generalized resistance to P. sorghi. Differences among rust ratings of the 40 inbred lines were highly significant (Table 29). No seasonal differences for rust ratings were found. C.V. for rust ratings over four seasons was 9.6%.

Table 28. Average rust ratings of 40 inbred lines planted on four different planting dates

Inbred	Planting date: Feb. 2 1972	Oct. 25 1972	Mar. 9 1973	Aug. 16 1973	Avg.
Oh545	1.2[+]	1.8	1.3	1.5	1.45
CM105	1.5	2.2	1.8	1.6	1.78
CM111	2.5	2.6	2.9	2.7	2.68
N6G	3.5	2.6	2.5	2.5	2.78
Ant2D	2.8	3.5	2.6	2.4	2.83
Oh43	3.0	3.0	2.9	4.0	3.23
Mo17	3.5	3.4	3.0	3.1	3.25
CI21E	3.5	2.2	3.7	3.9	3.33
Va26	3.4	3.4	3.2	3.5	3.38
M14	3.5	3.5	3.6	3.5	3.53
N28	3.5	3.7	2.8	4.5	3.63
38-11	4.0	4.0	3.6	3.5	3.78
B73	3.9	3.7	3.6	4.0	3.80
W64A	4.0	4.5	3.3	4.0	3.95
CI64	4.0	3.2	4.5	4.5	4.05
B68	5.0	3.6	4.4	5.5	4.63
A619	5.0	5.5	4.0	5.0	4.88
H95	5.0	5.1	4.7	4.9	4.93
Oh51A	5.0	4.8	5.1	5.0	4.98
B70	5.0	5.0	4.4	6.0	5.00
H55	5.0	4.8	5.4	5.5	5.00
H84	5.3	4.7	5.1	6.0	5.17
H60	5.5	5.2	6.0	5.6	5.28
F6	5.4	5.8	5.7	5.5	5.58
Mo18W	5.8	4.6	4.6	5.0	5.60
C123	6.0	5.7	5.8	5.3	5.70
C103	6.5	5.4	5.5	6.0	5.85
A632	5.5	5.3	6.4	6.7	5.98
Oh07	6.0	5.9	6.5	5.5	5.98
CI66	5.0	6.7	7.0	5.5	6.05
Va35	6.0	6.2	6.2	6.0	6.10
F44	6.0	5.6	7.0	6.0	6.15
Mo5	6.0	6.1	6.9	5.8	6.20
H94	6.0	6.1	7.0	6.0	6.28
CM104	6.5	6.0	6.7	6.2	6.35
AA8	7.0	5.9	6.8	6.3	6.50
B14	6.3	6.5	6.5	6.8	6.53
H93	6.8	6.6	7.0	6.0	6.60
B37	6.5	6.8	7.0	7.0	6.83
PH9	6.9	6.8	7.0	7.0	6.93
Avg.	4.70	4.82	4.85	4.88	4.81
BLSD at 5%					0.57

[+]Scale used was 1-7, ratings taken 4 weeks after mid-silking.

Table 29. Analysis of variance of data in Table 28

Source	d.f.	Mean Square
Inbred	39	8.630**
Seasons	3	0.254
Error	117	0.214
Total	159	

**P $<$ 0.01

The severe rust epiphytotics prevalent at the Waimanalo Farm appear to have permitted a more thorough examination of the resistance in maize lines than previously reported. Year-round corn culture and sprinkler irrigation enhance the rust epiphytotics in our maize nurseries. Possibly with more intensive corn culture in other wet subtropical regions, P. sorghi could become severe if generalized rust resistance genes are not incorporated as an objective in corn breeding programs for such regions. For the more humid wet areas of the U. S. Corn Belt, it may be important to utilize more extensively the generalized resistance inbred lines identified in present experiments and others in order to avoid potential damage that may be due to P. sorghi in certain favorable years.

4. Ontogenetic Changes of Generalized Resistance

It was considered important to determine if generalized resistance changes with plant growth stage and if such resistance could be detected prior to pollination.

Forty-two maize inbred lines planted March 8, 1973, were scored weekly for their generalized resistance based on a severity scale 1 to 7 from three weeks before mid-silking to four weeks after mid-silking. Average rust ratings of the 42 inbred lines are summarized in Table 30 and representative samples are graphically expressed in Figures 6, 7, and 8.

All resistant inbreds Oh545, CM105, CM111, and N6G showed a tendency toward resistance at one week before mid-silking, and continued to exhibit this ontogenetic change until two weeks following mid-silking (Fig. 6). Resistance in Oh545 apparently by lack of increase of lesion numbers and lack of increase of combined with expression of healthy tissue. No further infections were observed from mid-silking to the termination of the experiment. Sporulation also appeared to be somewhat inhibited. Resistance in CM105 developed dramatically between two weeks and one week prior to mid-silking. This was emphasized by increased plant vigor and development of new plant tissue which remained free from rust throughout the remaining experiment. The expressions of the high generalized resistance at one or two weeks before flowering in these inbreds suggest that much selection can be made prior to pollination.

Inbreds showing an intermediate reaction (CI21E, B73, M14, 38-11) had a tendency to fluctuate between susceptibility and mild resistance (Fig. 7). However, pustules continued to appear up to two weeks after mid-silking, and from this time on no new lesions were observed.

Susceptible inbreds B37, AA8, C123 and H84 developed lesions continuously (Fig. 8). Highly susceptible inbreds (B37, AA8) showed an infected area greater than 40% of the total leaf area prior to mid-silking. Plant growth was obviously suppressed by rust development. Moderately

Table 30. Weekly rust ratings of 42 inbred lines from mid-silking date (Scale 1-7).

	Weeks from mid-silking							
Inbred	-3	-2	-1	0	1	2	3	4
Oh545	4.1	3.8	3.0	1.7	1.5	1.3	1.3	1.3
CM105	4.8	4.6	3.0	2.5	2.4	2.0	1.9	1.8
CM111	4.9	4.8	4.1	3.9	3.3	3.0	2.9	2.9
N6G	4.7	4.4	3.9	3.7	3.1	2.9	2.5	2.5
Oh43	4.8	4.1	3.8	3.2	3.1	3.0	2.8	2.9
Mo17	4.7	4.1	3.6	3.7	3.5	3.0	3.0	3.0
CI21E	3.7	4.2	4.9	4.4	3.7	3.7	3.7	3.7
Va26	4.9	4.5	3.9	3.8	4.0	3.2	3.2	3.2
M14	3.8	4.0	4.0	3.6	3.6	3.5	3.6	3.6
N28	4.7	4.3	4.0	3.3	3.1	2.8	2.8	2.8
38-11	5.2	5.5	5.9	5.8	4.4	3.9	3.6	3.6
B73	4.7	4.8	4.6	4.5	3.7	3.8	3.6	3.6
W64A	4.5	4.0	3.3	3.0	3.3	3.3	3.3	3.3
Ky226	3.6	4.3	4.1	4.1	4.4	3.8	3.7	3.7
CI64	4.6	4.9	5.0	5.2	5.3	4.9	4.5	4.5
B68	4.5	4.4	5.8	5.9	5.8	4.4	4.4	4.4
A619	4.5	4.8	4.1	4.2	3.7	3.1	3.2	3.2
H95	4.9	4.8	5.3	5.2	4.9	4.6	4.7	4.7
Oh51A	4.6	4.8	4.9	5.0	5.2	5.3	5.1	5.1
B70	4.7	5.0	5.8	5.6	4.3	4.1	3.6	3.6
H55	5.0	4.5	4.3	4.5	4.3	4.1	3.5	3.4
H84	4.9	4.9	5.1	5.4	5.6	5.3	5.4	5.4
H60	5.0	4.7	5.6	5.5	4.8	5.0	5.1	5.1
F6	3.5	5.4	5.5	5.4	5.7	5.9	6.0	6.0
Mo18W	3.9	4.7	5.8	6.1	6.2	6.5	6.7	6.7
C123	5.0	5.1	5.7	5.6	5.7	5.7	5.8	5.8
C103	5.0	5.1	5.9	5.9	5.8	5.6	5.5	5.5
A632	5.0	5.1	5.0	5.9	6.1	6.3	6.3	6.3
Oh07	4.9	5.0	5.3	5.5	5.9	5.9	6.3	6.5
CI66	4.6	5.0	6.8	7.0	7.0	7.0	7.0	7.0
Va35	4.3	4.6	6.0	5.9	6.2	6.2	6.2	6.2
F44	6.0	6.9	7.0	7.0	7.0	7.0	7.0	7.0
Mo5	5.0	5.0	5.8	6.2	6.5	6.8	6.9	6.9
H94	5.7	5.9	6.9	6.9	7.0	7.0	7.0	7.0
CM104	4.8	5.0	5.1	6.3	6.4	6.4	6.4	6.7
AA8	5.8	5.9	6.7	6.9	7.0	7.0	7.0	7.0
B14	4.5	5.0	5.0	6.8	7.0	7.0	7.0	7.0
H93	5.5	6.7	6.9	7.0	7.0	7.0	7.0	7.0
B37	5.0	5.5	6.6	6.9	6.9	7.0	7.0	7.0
T232	5.0	5.0	6.9	7.0	7.0	7.0	7.0	7.0
PH9	5.3	5.8	6.0	6.9	7.0	7.0	7.0	7.0
Ga209	5.2	5.1	6.1	7.0	7.0	7.0	7.0	7.0

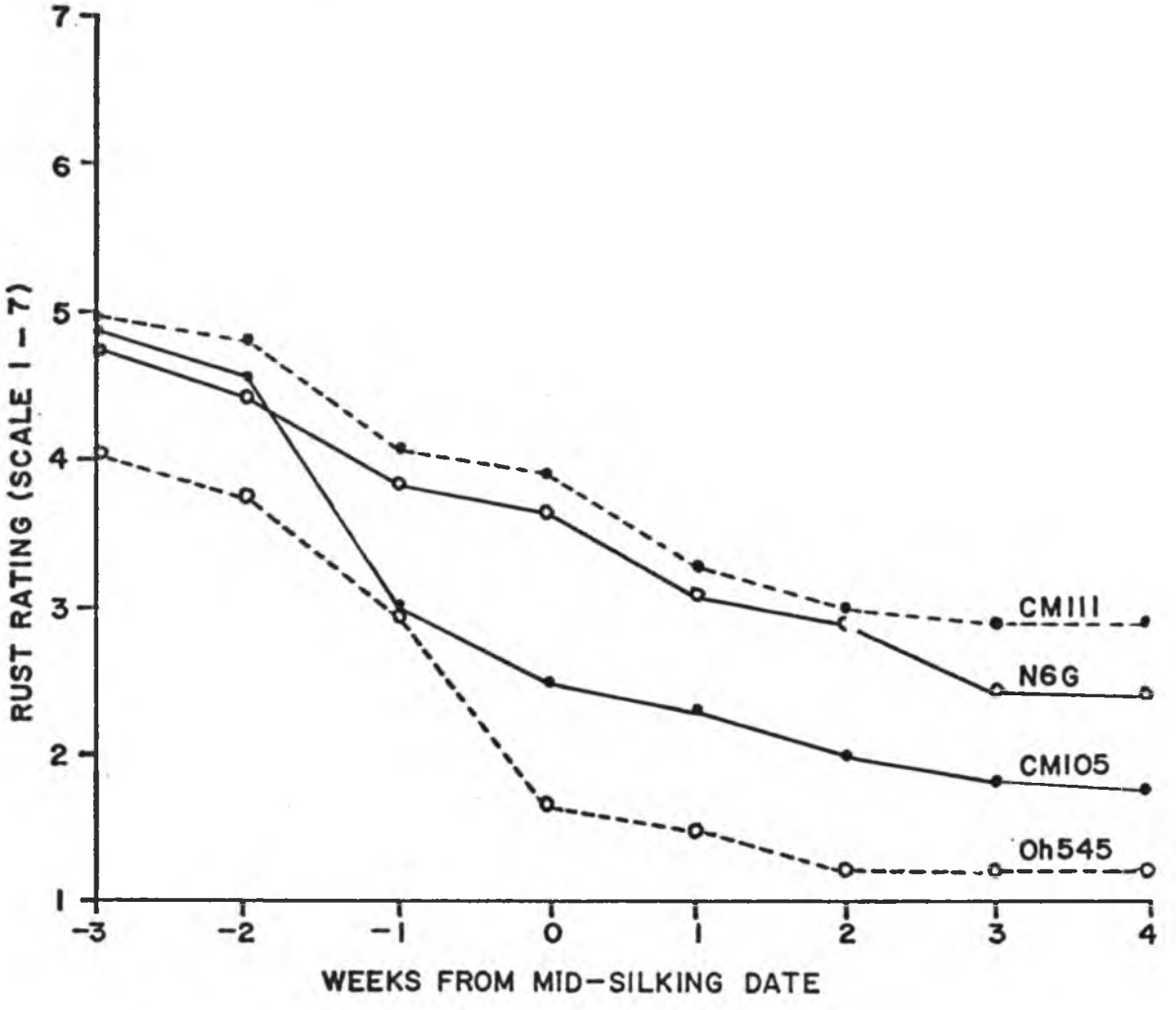


Figure 6. Weekly changes in rust ratings of 4 generalized resistant inbreds (weeks from mid-silking date).

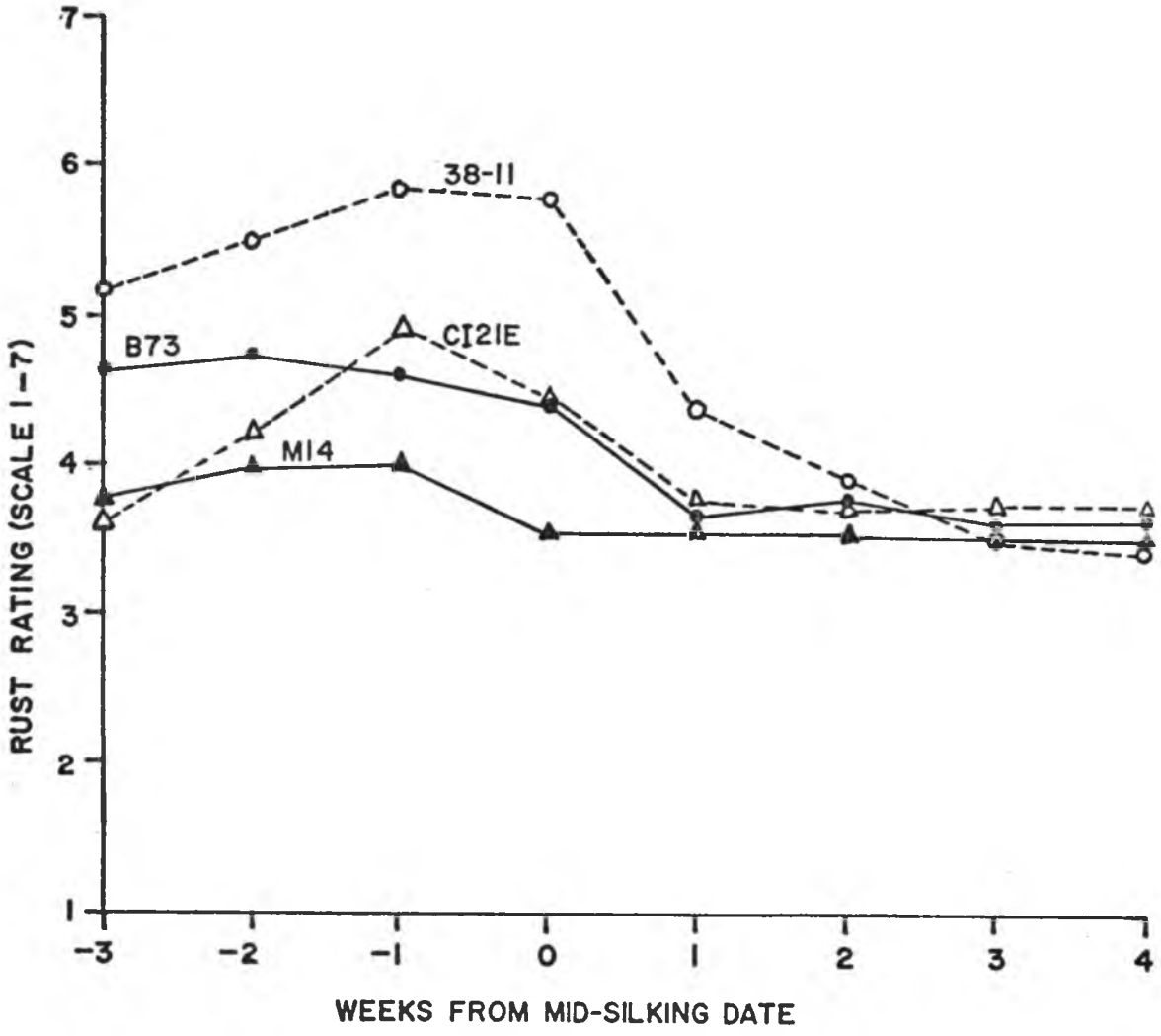


Figure 7. Weekly changes in rust ratings of 4 intermediate inbreds (weeks from mid-silking date).

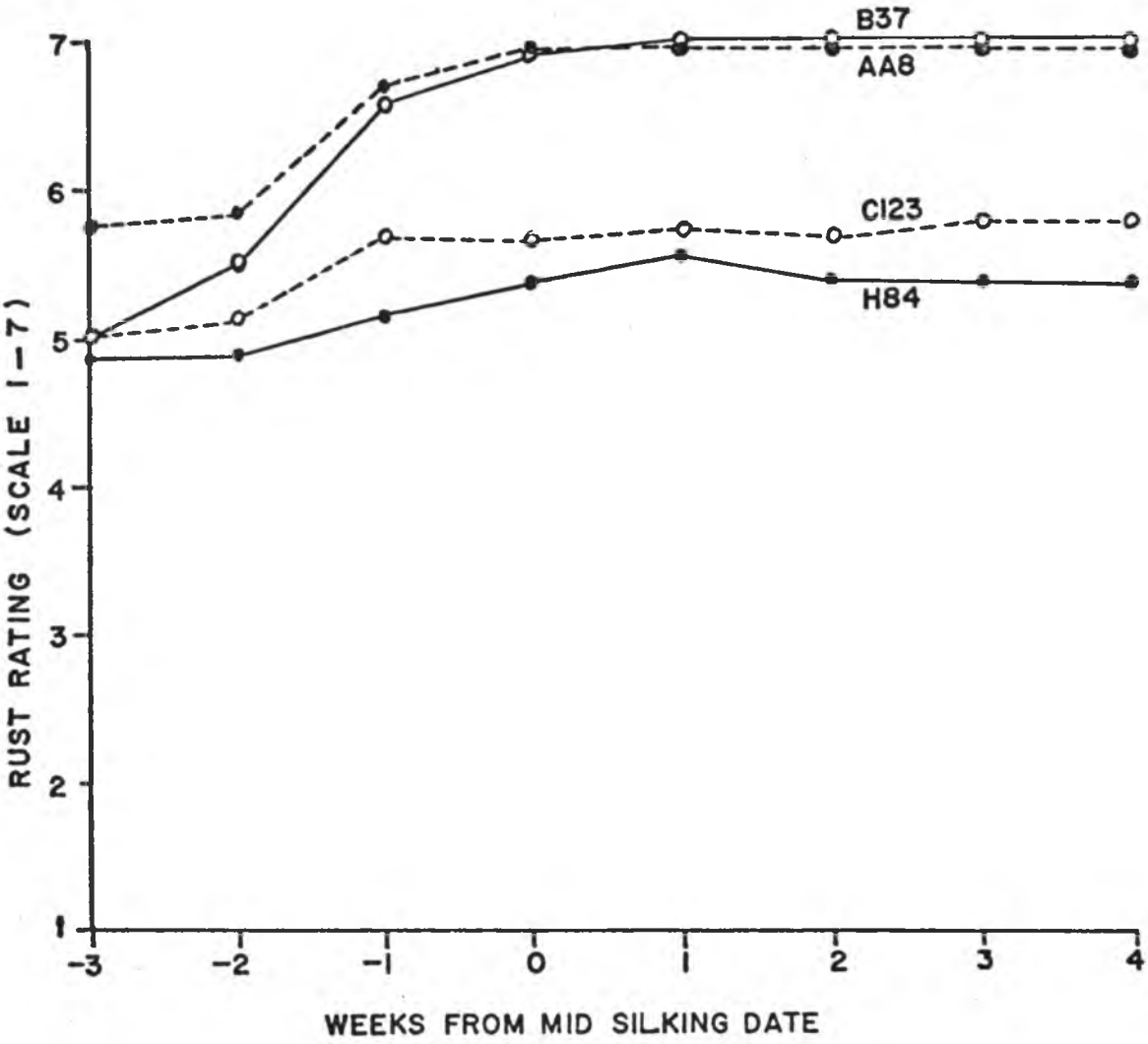


Figure 8. Weekly changes in rust ratings of 4 susceptible inbreds (weeks from mid-silking date).

susceptible inbreds (C123, H84) evidenced growth throughout the experiment, however, growth was significantly retarded.

5. Susceptibility of Maize Races

The 41 tropical maize races varied considerably in rust reactions (Table 31). Five of the 41 tested races had a fleck-type response which may be controlled by specific resistance genes. However, genetic studies would be required to establish the nature of these reactions. Seven of the 36 non-flecking races were considered to have good generalized resistance and 15 were considered to have intermediate level of generalized resistance. Eight of the 36 were considered fully susceptible and six races were considered intermediate between resistance and susceptibility. Wellhausen et al. (1952) observed large differences in the reactions among 25 maize races to P. sorghi. Among these 25 races, Harinoso de Ocho rated as one of the best resistant races, but it was highly susceptible in Hawaii (Table 31). Hooker and LeRoux (1957) obtained a majority of the fleck-type resistance sources from Mexican maize lines. Some of the classifications of rust reactions in present results may be incorrect, because many of the races were heterogeneous for rust reactions.

6. 677a Resistance

Preliminary observations of rust resistance in sweet corn inbred 677a were made in 1972. Individual F_2 plants derived from (677a X AA8) segregated 211 susceptible to 74 resistant. This result suggested that 677a resistance was controlled by a single recessive gene.

It was generally observed that 677a showed susceptible symptoms on the first several leaves, and later produced unusual reddish-purple flecking symptoms on the leaves (Fig. 9). Sporulation was clearly reduced at this stage, with some sporulation at the center of the flecks.

Table 31. Arbitrary grouping of 41 maize races based on rust reactions over two seasons

Complete resistance (fleck-type):

Amagaceno, Bolita 2822, Cacao amarillo, Chulpi, and Cabuya amarilla.

High generalized resistance (Rust score range: 1.4-2.0):

Amagaceno blanco (1.6), Caingang (2.0), Clavo (1.4), Comiteco mezcla (1.8), Imbricado blanco (1.7), Palomero Toluqueno (1.4), and Sabanero blanco (2.0).

Moderate generalized resistance (Rust score range: 2.2-3.6)

Amagaceno amarillo (2.9), Andaqui (2.9), Avati-Tupi (2.9), Cariaco (2.6), Chapalote 2833 (2.9), Chococeno (3.6), Comun amarilla (3.0), Conico norteno (2.2), Guirua (2.6), Montano blanco (3.0), Nal-Tel (3.5), Sabanero (3.3), Tehua (2.8), Tuxpeno (3.0), and Vandeno (3.6).

Intermediate between resistant and susceptible (Rust score range: 4.0-4.6):

Calchaqui (4.6), Chococeno cristalino blanco (4.0), Guiru mezcla (4.0), Lady Finger pop (4.8), Negrito (4.9), and Zapalote Grande (4.2).

Susceptible (Rust score range: 5.0-5.3):

Comun blanco (5.0), Coastal Tropical flint (5.0), Costeno amarillo (5.3), Cuban flint (5.0), Haitian yellow (5.2), Harinoso de Ocho (5.0), Puya (5.2), and Yucatan (5.0).

A total of 1856 plants was classified for rust reactions (Table 32). None of the F_1 plants showed 677a resistance symptoms (Fig. 9). The F_2 and backcross segregations clearly indicated that 677a resistance is controlled by a single recessive gene, allelic to Rp_1^d. Average X^2 value for the three F_2 population was 0.278, and the average probability value was over .75. Average X^2 value for the backcross population was 0.063, with P 0.80.

Although some variations in 677a resistant symptom expression such as lesion size occurred in segregating generations, the results obtained in this study suggested that 677a resistance is controlled by a single recessive allele of the Rp_1 locus. Rust resistances controlled by recessive gene(s) have been reported (Hooker and LeRoux 1957, Malm and Hooker 1962). In the resistant lines Amargo 47 and Midland 125, high resistance was controlled by three multiple recessive genes rpa, rpb, and rpc (Malm and Hooker 1962).

Table 32. Reaction of F_1, F_2, and backcross populations to *P. sorghi*

	Cross	Observed No. Res.	Observed No. Sus.	Expected No. Res.	Expected No. Sus.	X^2	P value
F_1	677a X AA8b	0	105				
	677a X 442	0	102				
	677a X Rp_1^d	677a res. 0	Rp res. 120				
F_2	(677a X AA8b)	163	486	162.25	486.75	0.005	.90-.95
	(677a X 442)	67	177	61.00	183.00	0.787	.70-.80
	(677a X Rp_1^d)	677a res. 47	Rp res. 146	48.25	144.75	.043	.80-.90
BC	(677a X AA8b) 677a	143	138	140.5	140.5	0.089	.70-.80
	(677a X 442) 677a	25	26	25.5	25.5	0.920	.80-.90
	(677a X Rp_1^d) 677a	677a res. 54	Rp res. 57	55.5	55.5	0.081	.70-.80

Figure 9. Phenotypic expressions of hybrid 677a X AA8 and their respective parents at mid-silking.

III. ESTIMATES OF GENERAL AND SPECIFIC COMBINING ABILITY ON CORN INFECTED WITH *PUCCINIA SORGHI* (DIALLEL ANALYSIS)

ABSTRACT

Nine parents and their 36 F_1 hybrids of maize (*Zea mays* L.) were evaluated for the inheritance of resistance to *Puccinia sorghi* rust by means of diallel analyses. Highly significant differences in rust ratings were detected among the 45 genotypes. Two inbreds, Oh545 and CM105, were outstanding in resistance and in their high GCA (general combining ability) for resistance. Highly significant GCA and SCA (specific combining ability) mean squares were obtained, with GCA always substantially larger than SCA. Broad sense heritability was 85%, and narrow sense heritability 72%, and environmental effects were extremely small ($<1\%$). A high coefficient of linear determination ($r^2=.91$) was calculated between parental averages and GCA effects. Since additive genetic variance accounts for a major portion of the genotypic variance for rust resistance, it should not be difficult to increase resistance by traditional breeding methods. Selected SCA effects appeared large enough, however, to encourage attention to the possibility that certain hybrids may be more resistant than expected on the basis of GCA values.

MATERIALS AND METHODS

Nine corn lines with a wide range in resistance to rust were selected for this study. Five of the lines (Ant2D, CM105, CM111, Mo17, and Oh545) had previously shown high or moderate generalized resistance to P. sorghi in inbred screening tests (Table 28). One line (CI21E) was classified as intermediate and the remaining three lines (AA8, B37, and CM104) were classified as highly susceptible. All possible F_1 crosses were made in 1971 and 1972. The nine parents and 36 F_1 progenies were planted under severe epiphytotics at the Waimanalo Experiment Farm, University of Hawaii, during June and September in 1973.

A randomized complete block design with four replications was employed. Each plot had 15 plants spaced 20 cm between plants and 75 cm between rows. Susceptible hybrids were planted at the end of each row. Ten individual plants per row were scored for rust three weeks after mid-silking, rating the whole leaves on a 1 to 7 scale for percentage of leaf area covered with rust. The 7 point scale system was modified from the rating scales for leaf rusts of cereals proposed by Peterson et al. (1946) and James (1972). P. sorghi urediospore suspension obtained from infected healthy leaves of susceptible plants in the previous crop were sprayed only on the susceptible hill plants by a five-gallon Knapsack sprayer at 7 to 8 leaf stage. Sprinkler irrigations were applied approximately twice a week during the experiment. After the inoculation on July 15th, 1973, weekly relative humidities in excess of 95%, and minimum and maximum temperatures were recorded (Fig. 10).

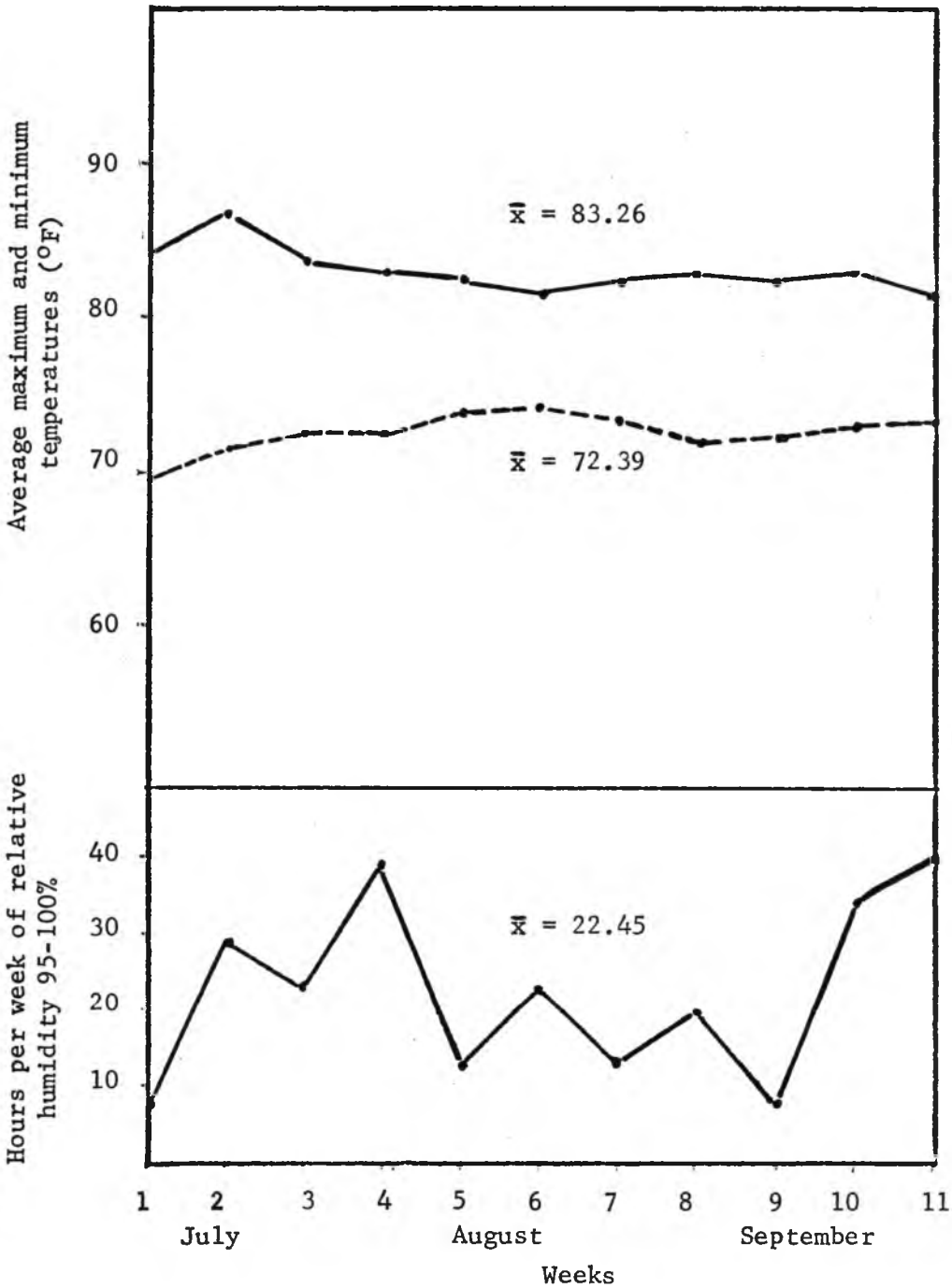


Figure 10. Weekly relative humidities in excess of 95%, and minimum and maximum temperatures in the corn field at the Waimanalo Farm.

Griffing's (1956) methods 2 and 4, and models I and II for diallel analyses were employed to estimate general and specific combining abilities and variance and covariance components based on the individual rust ratings from 10 plants per plot.

The computation facilities of the University of Hawaii Statistical Computing Center were employed. All data were processed according to the programs of Littlewood et al. (1964). Griffing described 4 experimental methods of analyzing and interpreting data from diallel crosses; The four methods are:

Method 1 utilizes parents, F_1's and their reciprocals.

Method 2 utilizes parents and F_1's, excluding reciprocals.

Method 3 utilizes F_1's and reciprocals, excluding parents.

Method 4 utilizes F_1's, excluding parents and reciprocals.

In model I ("Fixed Model"), parents are chosen as a fixed sample, and data can be used to estimate GCA and SCA effects and to compute standard errors for differences between effects. In model II ("Random Model"), parents are believed to represent a random sample from the population of inbreds, and data can be used to estimate genotypic and environmental components of the population variance, and to estimate the standard errors of these components. These analyses are valid only if genotype mean squares are highly significant.

RESULTS

1. Rust Ratings of 9 X 9 Diallel

Heavy rust epiphytotics were achieved by planting and inoculating susceptible hybrids as hill plots adjacent to the experimental materials. The year-round corn culture and sprinkler irrigation system enhances rust epiphytotics at the Waimanalo Farm, often markedly reducing grain yields in this area (Table 4).

Nine parents and their 36 F_1 hybrids were scored for rust under this epiphytotic. Rust ratings based on the scale of 1 to 7, 1 being highly resistant and 7 being highly susceptible, are summarized in Table 33. Parent, F_1, and grand means were 3.82, 3.83, and 3.83 respectively. Wide differences for rust ratings were found in both parents and F_1. The range of the nine parents was from 1.18 to 6.48, and that of the 36 F_1 hybrids was from 1.10 to 6.43.

Five parental lines, Oh545, CM105, Ant2D, CM111, and Mo17 showed some degree of generalized resistance to rust (Table 33). The best resistance was that of yellow dent inbred Oh545, which was developed cooperatively by the Ohio Agricultural Research and Development Center and U. S. Department of Agriculture and released to corn breeders in 1970. Inbred Oh545 was derived from [(M14 X CI187-2) Oh45 X Oh45] X [(Oh45T4 X Cash) X M14 X CI187-2) Oh45)] X Oh45A

CM105 was bred in India by prolonged sib-pollination from a Peruvian variety, Peru 330, and was second in rust resistance in this experiment.

Three parents, CM104, AA8, and B37 were highly susceptible to rust. B37, one of the most widely used U. S. Corn Belt dent inbred lines, was the most susceptible. AA8, a Hawaiian sweet inbred and CM104, a

Table 33. Rust ratings of 9 parents and their 36 F_1 hybrids*

Parent	Oh545	CM105	Ant2D	CM111	Mo17	CI21E	CM104	AA8	B37	Avg. of Hybrid
Oh545	1.18	1.10	1.98	1.95	2.28	2.68	2.38	2.98	2.63	2.25
CM105		2.30	2.80	2.20	3.35	3.00	3.55	4.03	3.70	3.23
Ant2D			2.55	4.73	3.45	4.13	4.05	3.10	5.18	4.11
CM111				2.55	3.63	4.55	3.75	3.93	4.95	4.16
Mo17					2.98	3.98	4.35	3.98	4.28	4.15
CI21E						4.15	4.98	5.70	6.00	5.56
CM104							6.20	5.90	6.43	6.17
AA8								6.25	6.20	6.20
B37									6.48	

*Grand mean = 3.83, Parental mean = 3.82 and F_1 mean = 3.83.

Average heterotic effect in per cent = 2.3.

se_d of treatment means based on parent and F_1 data = 0.083.

se_d of the difference between two treatment means = 0.118.

yellow Colombian flint bred in India were also highly susceptible. CI21E was intermediate in resistance.

The pattern of resistance among F_1 hybrids appeared to follow the expectation that inheritance was polygenic with relatively high general combining ability for resistance. The ranking of hybrids (Table 33) closely followed that of the common parent. The progeny of Oh545 were outstanding for their resistance, always exceeding that of the susceptible parent. All hybrids of the 3 most susceptible inbreds showed no more resistance than the parent inbreds, despite impressive hybrid vigor.

Heterotic effects in percent were calculated from individual F_1 hybrid values over their respective mid-parent values and averaged only 2.3%, indicating that hybrid vigor did not influence maize resistance to P. sorghi in these materials (Table 33).

2. Analyses of Variance and Covariance

Mean squares from the randomized block analysis of variance for parental and hybrid rust ratings are summarized in Table 34, based on Griffing's methods 2 and 4 and fixed model I (Griffing 1956). Highly significant differences were detected among genotypes for rust ratings in both methods, a necessary condition for computation of the combining abilities summarized in Table 35. Variations due to GCA and SCA were both highly significant from a variance components analysis of rust ratings. GCA and SCA effects accounted for 73.4 and 26.1% in method 2, and 75.1 and 24.3% in method 4 (Table 36).

Environmental variances were extremely small, accounting for 0.56% of total variance for method 2 and 0.58% for method 4, respectively (Table 36). The results indicated that replication was adequate, and

Table 34. Analyses of variance of rust ratings in Table 33 based on Griffing's methods 2 and 4, fixed model I

Source	Method 2 (Parental and F_1)		Method 4 (Only F_1)	
	d.f.	Mean square	d.f.	Mean square
Genotype	44	82.436**	35	67.619**
Replication	3	16.007	3	18.601
Genotype X rep	132	2.576	105	2.682
Error	1620	0.278	1296	0.279

**$p < 0.01$

Table 35. Combining ability mean squares based on methods 2 and 4, model I, for rust ratings

Source	Method 2		Method 4	
	d.f.	Mean square	d.f.	Mean square
GCA	8	10.021**	8	6.377**
SCA	36	0.330**	27	0.302**
Error	1620	0.007	1296	0.007

**$p < 0.01$

Table 36. Variance components analysis in per cent for GCA and SCA values in Table 35

Component	Method 2		Method 4	
	Variance	Percent	Variance	Percent
GCA	0.910	73.4	0.910	75.1
SCA	0.323	26.1	0.295	24.3
Error	0.007	0.6	0.007	0.6

suggested little need for repetition of the experiment.

On the random model, GCA and SCA mean squares were also highly significant, based on error mean square of 0.064 (Method 2) and 0.067 (Method 4) respectively. Application of the random model to these data was made on the assumption that the parents represent a truly random sample of maize inbreds in terms of rust resistance.

Estimates of total phenotypic variance (Vp), total genotypic variance (V_G), additive genetic variance (V_A), nonadditive genetic variance (V_d), and environmental variance (V_E), as well as broad (bH) and narrow (nH) sense heritabilities are summarized in Table 37. The genotypic variances were approximately 2.0 for both methods, accounting for a major portion of the phenotypic variances of 2.3. Additive genetic variances were 1.76 and 1.74 for method 2 and 4 respectively, and constituted a major portion of the total genotypic variance. Non-additive genetic variances were only .27 and .24 for the two methods and constituted a very small portion of the total genotypic variance.

Broad and narrow sense heritabilities were calculated, using the following notations:

$$bH = V_G / V_p$$

$$nH = V_A / V_p$$

The broad sense heritabilities were 85.8% for method 2 and 85.1% for method 4, and narrow sense heritabilities were 72.1% and 72.7%, respectively. High narrow sense heritability estimates reflect the high additive gene action in the inheritance of rust resistance in maize (Table 37).

3. General Combining Ability Effects

General combining ability effects for the nine parents are summarized

Table 37. Estimates of phenotypic variance and its components, and of broad (bH) and narrow (nH) heritabilities for rust ratings

	Method 2	Method 4
V_P	2.306 ± 0.915	2.250 ± 0.915
V_G	2.028 ± 0.915	1.971 ± 0.915
V_A	1.762 ± 0.911	1.736 ± 0.911
V_D	0.266 ± 0.078	0.235 ± 0.083
V_E	0.278 ± 0.010	0.179 ± 0.011
bH	85.8 (80.1-91.4)	85.1 (79.1-91.2)
nH	72.1 (61.2-83.0)	72.7 (61.8-83.6)

Table 38. Estimates of general combining ability effects for rust ratings

Parent	Parental mean	General combining ability effects	
		Method 2	Method 4
Oh545	1.18	-1.63	-1.81
CM105	2.03	-0.95	-0.99
Ant2D	2.55	-0.34	-0.19
CM111	2.55	-0.32	-0.17
Mo17	2.98	-0.28	-0.14
CI21E	4.15	0.46	0.63
CM104	6.20	0.87	0.68
AA8	6.25	0.91	0.71
B37	6.48	1.28	1.25
se_d		0.036	0.045

in Table 38, as calculated on the fixed model. As noted for the original data (Table 33), GCA effects differed widely among the parents. Five parents-Oh545, CM105, Ant2D, CM111, and Mo17 in decreasing order--had highly significant negative GCA effects, indicating that these five parents were good general combiners and increased resistance in hybrid combinations. The four remaining parents-B37, AA8, CM104, and CI21E had significant positive GCA effects, indicating that these parents were poor general combiners for resistance to rust and increased susceptibility. Inbred Oh545, on the average, reduced rust symptom expression by 1.6 (method 2) or 1.8 (method 4), on the rust rating scale of 1 to 7 scale, in hybrid combinations with the remaining eight parental inbreds. By contrast, the genetic contributions of inbred B37, on the average, increased rust susceptibility by 1.3 (method 2) and 1.2 (method 4) in hybrid combinations.

The coefficient of linear determination (r^2=0.91**) between parental averages and their GCA effects based on method 2 for rust ratings was extremely high, and this was highly significant.

4. Specific Combining Ability

Specific combining ability effects based on methods 2 and 4 for rust ratings are summarized in Table 39. SCA effects represent deviations of an individual F_1 hybrid from an average based on its parental GCA effects. There was little difference between individual SCA effects calculated by the two methods. Hybrid Ant2D X AA8 showed the highest SCA effect for increasing resistance in hybrid combination, ranking 1.3 units more resistant than expected on the basis of the additive contributions of the parental lines evaluated. CM111 X Ant2D showed the lowest SCA effect, ranking 1.6 unit (method 2) less resistant

Table 39. Estimates of specific combining ability effects for rust ratings (Method 2 results above diagonal; Method 4 below)

Parent	Oh545	CM105	Ant2D	CM111	Mo17	CI21E	CM104	AA8	B37
Oh545		-0.14	0.12	0.07	0.36	0.02	-0.68	-0.13	-0.85
CM105	0.07		0.27	-0.36	0.75	-0.33	-0.19	0.24	-0.45
Ant2D	0.13	0.13		1.56	0.24	0.18	-0.30	-1.30	0.41
CM111	0.07	-0.51	1.21		0.39	0.58	-0.63	-0.50	0.16
Mo17	0.45	0.70	-0.01	0.12		-0.03	-0.07	-0.49	-0.55
CI21E	0.03	-0.47	-0.15	0.23	-0.29		-0.18	0.50	0.44
CM104	-0.32	0.02	-0.28	-0.62	0.03	-0.16		0.30	0.46
AA8	-0.22	0.44	-1.30	-0.51	-0.40	0.51	0.65		0.18
B37	-0.64	-0.39	0.27	0.01	-0.61	0.30	0.67	0.38	

se_d between effects of two parent lines based on Method 2 = 0.094

se_d between effects of two crosses having one parent line in common:

Method 2: 0.112

Method 4: 0.109

se_d between effects of two crosses having no parent lines in common:

Method 2: 0.107

Method 4: 0.100

than expected on the basis of the additive genetic contribution. Five crosses with CM105 showed negative SCA effects from both methods, indicating that these crosses were also better than expected on the basis of the average performance of the parental lines involved for rust resistance. Six individual crosses with B37 and CI21E showed positive SCA effects, indicating that these crosses were worse than expected on the basis of their average performance.

DISCUSSION

Diallel crosses used in this study were analyzed using both method 2 (parents and F_1) and method 4 (F_1 only) and both fixed model I and random II (Griffing 1956). The results from Griffing's method 2 and 4 analyses were almost identical, suggesting that heterosis does not influence rust ratings in hybrid combinations. Griffing (1956) generally recommended method 4, utilizing F_1 crosses and excluding parents, for quantitative genetic studies in plant breeding, as a result of the widespread occurrence of heterosis. In the case of P. sorghi resistance, inclusion of the parents (method 2) is clearly warranted.

Highly significant differences for rust ratings occurred among the 9 different inbreds, supporting earlier results of inbred line tests in four different seasons (Table 28). Oh545 and CM105 were found to be excellent of sources resistance.

General combining ability estimates for rust resistance were substantially larger than specific combining abilities, and narrow sense heritability was very high, 72%. High general combining ability effects have been reported for Maize Dwarf Mosaic Virus (Josephson and Naidu 1971, Johnson 1971, Loesch and Zuber 1972) and corn stunt (Nelson and Scott 1973). Loesch and Zumber (1972) studied 45 single crosses for the inheritance of MDMV resistance in maize, using Griffing Method 4 (utilizing only F_1's). Results indicated that both GCA and SCA mean squares were significant, but the GCA mean square was substantially larger than the SCA man square. Greater resistance was observed in F_1 hybrids when compared with parental lines. They concluded that diallel

analysis with only F_1 crosses, was a superior procedure for estimating variance components in MDMV resistance in maize. Similar results also reported in MDMV resistance (Josephson and Naidu 1971, Johnson 1971). Nelson and Scott (1973) evaluated 45 single crosses (10 inbred lines) of maize for resistance to corn stunt in maize. Results indicated that GCA mean squares were highly significant and were much larger than SCA mean squares for corn stunt resistance. They concluded that additive gene action appeared to be major contribution to the inheritance of resistance to corn stunt. A breeding program such as simple recurrent selection was recommended for developing new resistant inbred lines to corn stunt. High additive gene action in the inheritance of disease resistance to Northern corn leaft blight, caused by Helminthosporium turcicum, had been reported (Hughes and Hooker 1972). No report, however, has been made on the combining ability effects in P. sorghi as well as P. polysora resistance.

High additive genetic variance for rust resistance obtained in this study suggests that resistance can be improved effectively in genetically variable populations by breeding methods such as phenotypic recurrent or mass selection. Estimate of high coefficient of linear determination (r^2) between parental rust averages and their GCA effects suggests that corn breeder can select parental lines for resistance and utilize effectively their GCA values in his breeding programs.

Specific combining ability effects from 36 F_1 hybrids were of minor importance in the inheritance of rust resistance. In very few crosses, e.g., Ant2D X AA8, specific gene combinations or interactions produced much greater resistance than predicted on the basis of GCA values. The importance of the specific combining ability for MDMV

resistance has been reported by Josephson and Naidu (1971). It is doubtful that selection advance for rust resistance during inbreeding corn, for example, would be improved practically by a system that exploits both general and specific combining ability.

Approximately 85 percent of the total phenotypic variance for rust resistance was due to genetic causes, expressed as a high broad sense heritability. Hooker (1967, 1969) calculated very similar heritability estimates for rust resistance from 45 F_2 populations. The most promising way of maintaining P. sorghi as a minor economic corn disease in the long run may be by exploiting generalized resistance.

IV. GENERALIZED RESISTANCE TO P. SORGHI IN MAIZE DESCRIBED GENETICALLY BY GENERATION MEAN ANALYSIS

ABSTRACT

Quantitative genetic studies of resistance in maize to Puccinia sorghi Schw. were made in the 25 possible F_1 crosses between five resistant lines (Oh545, CM105, CM111, Mo17, and CI21E) and five susceptible lines (B37, AA8, CM104, C123, and H95) and in their corresponding F_2 and backcross progenies. Gene action was interpreted on a polygenic basis, assessing the importance of both additive and non-dominance genetic variances. Additive gene action was of major contribution for rust resistance in this experiment and accounted for 55.1% of the total genotypic variance. High additive variances were detected in crosses involving the three most resistant lines, Oh545, CM105, and CM111. High dominance or non-additive variances were observed primarily in crosses involving the two moderate resistant lines, Mo17 and CI21E. 47.0% narrow sense and 83.4% broad sense heritabilities were estimated for rust resistance in the 25 crosses.

A highly significant negative correlation (r=-.63) was detected between F_1 rust ratings and their corresponding additive variances. An expected genetic advance through selection of 26.9% at the 5% level was also estimated. The calculated average minimum numbers of gene pairs controlling resistance were 1.24, 0.92, and 0.94 for the F_2, B_r, and B_s progenies, respectively. These results agree well with the results obtained in previous studies based on diallel analyses. It is recommended that a breeding program for selection of lines or crosses that contribute polygenic rust resistance based on highly additive gene

action would be desirable to keep P. sorghi minor economic disease in maize.

MATERIALS AND METHODS

Ten maize inbreds were chosen for generation mean analyses of rust resistance, five showing varying levels of resistance and five quite susceptible. The characteristics of these 10 parents were as follows:

Parent	Kernel	Origin		Rust resistant score (Table 3)
Oh545	Dent	Ohio	Oh45 (X M14, CI187-2) etc.	1.45
CM105	Flint	India	Peru 330#	1.78
CM111	Flint	India	Cuba 342-2-2-#	2.68
Mo17	Dent	Missouri	CI187-2 X C103	3.25
CI21E	Flint	USDA-Indiana	K577C X $(Hy)^2$	3.33
H95	Dent	Indiana	Oh43 X CI90A	4.93
C123	Dent	Connecticut	C102 X C103	5.70
CM104	Flint	India	Amarillo Theobromina 21 (B) -f#	6.35
AA8	Sweet	Hawaii	Hawaii A19-6-1-1	6.50
B37	Dent	Iowa	Iowa Stiff-Stalk Synthetic	6.83

The 25 possible F_1 crosses between the 5 resistant and 5 susceptible parents and their respective 25 F_2 and 50 backcrosses (25 B_r and 25 B_s) were made at the Waimanalo Farm on Oahu, Hawaii in 1971 and 1972.

Two plantings were made, on August 1973 and on February 1974 at the Waimanalo Farm. Each planting contained 110 populations including 5 P_r, 5 P_s, 25 F_1, 25 F_2, 25 B_r, and 25 B_s.

Randomized complete block designs with four replications in 1973 and with three replications in 1974 were employed. Parents and F_1, F_2,

and both backcrosses plots were grouped together in each replication. All entries within a plot were randomized. A replication consisted of one-row plots of each parent and F_1, four-row plots of F_2, and three-row plots of each backcross. Each row had 15 plants.

In 1973 all plants were inoculated at 8 to 9 leaf stage by ground rust inoculum collected from the infected leaves of susceptible plants in the previous crop. In 1974 the inoculations were made only on the susceptibel spreader plants which were planted every 10 rows 2 weeks earlier than the experimental materials.

A total population of 20,250 plants was scored for rust ratings based on the scale 1 to 7 described previously, 1 being highly resistant and 7 being highly susceptible three to four weeks after mid-silking. Approximately 70 plants were scored in each parental and F_1 population, 305 plants in each F_2, and 204 plants in each backcross over two seasons.

Estimates of the additive (A) and dominance or non-additive (D) variance components, and of heritability (H) were obtained by the methods of Mather (1949). Assuming that the genes neither interact nor are linked, the F_2 progenies segregating from selfing of F_1 hybrids have the following phenotypic variance:

(1) $$VF_2 = \frac{1}{2}a^2 + \frac{1}{4}d^2 + V_E$$

Where $\frac{1}{2}a^2$ represents additive genetic variance (V_A), $\frac{1}{4}d^2$ represents non-additive genetic variance (V_D), and V_E represents environmental variance. Similarly the sum of the phenotypic variances of both backcrosses is represented by

(2) $$VB_r + VB_s = \frac{1}{2}a^2 + \frac{1}{2}d^2 + 2V_E$$

Other pertinent calculations are the following:

(3) $2 \times VF_2 - (2) = \frac{1}{2}a^2 = V_A$

(4) $V_E = \frac{VP_r + VP_s + VF_1}{3}$

(5) Using the variance for the F_2 population,

$VF_2 = V_A + V_D + V_E$, or $V_D = VF_2 - V_A - V_E$

Heritability in the narrow sense, relating additive to phenotypic variances was measured following Warner (1952), by using the components of Mather's equation:

$$nH = \frac{2VF_2 - (VB_r + VB_s)}{VF_2} = \frac{\frac{1}{2}a^2}{\frac{1}{2}^2 + \frac{1}{4}^2 + V_E}$$

Genetic advances through selection were calculated by the equation proposed by Comstock and Robinson (1952).

Minimum numbers of gene pairs controlling rust resistance were calculated in two ways by the equations proposed by Weber (1950), and Castle and Wright (1921).

Pooled rust ratings' data over two seasons with seven replications were used for these estimates described above.

Weekly humidities (RH) exceeding 95%, and minimum and maximum temperatures after the inoculation of spreader plants inside the corn field were recorded by hydrothermometer, and summarized in Figure 11 and Figure 12.

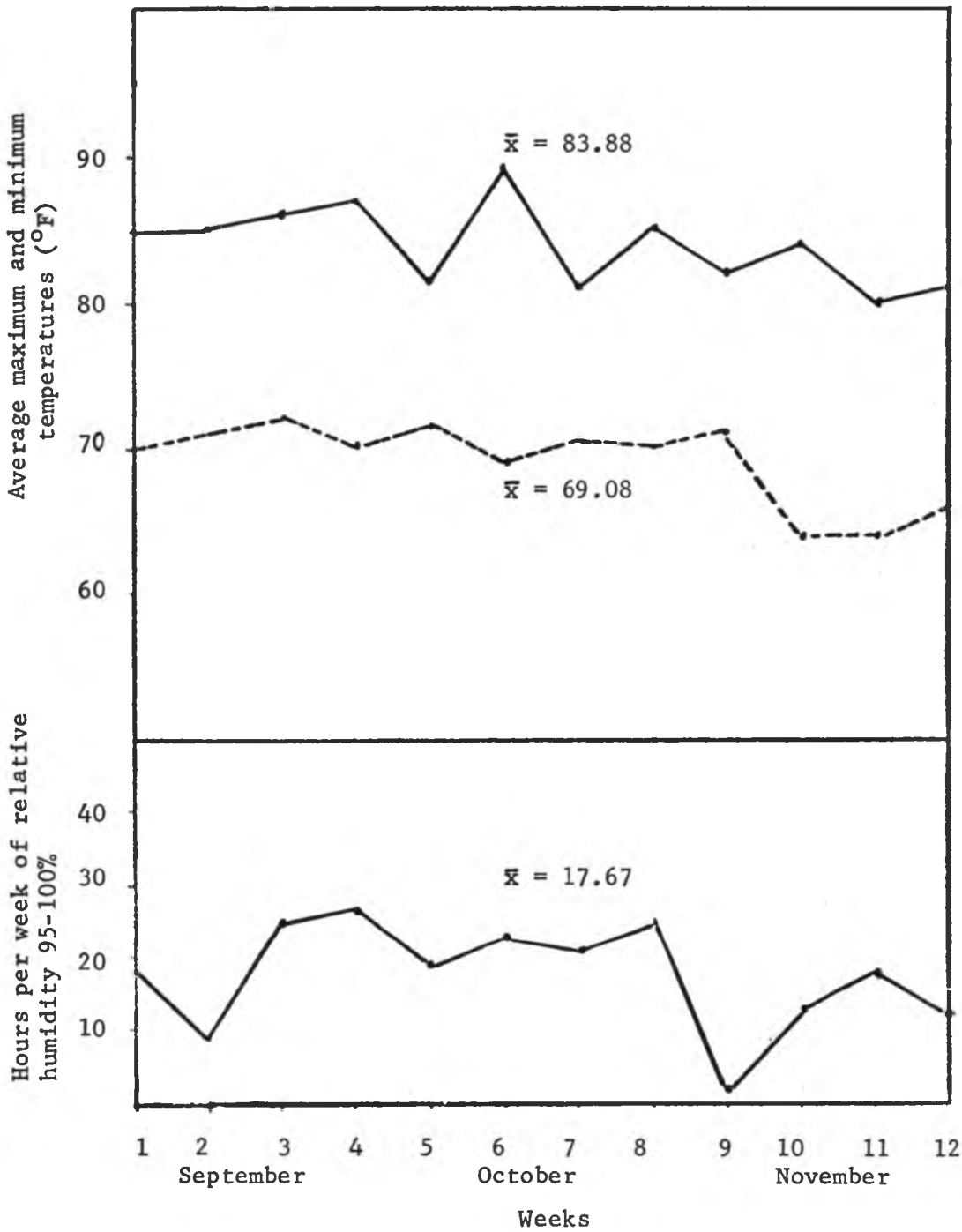


Figure 11. Weekly relative humidities in excess of 95%, and minimum and maximum temperatures in the corn field during September and November in 1973.

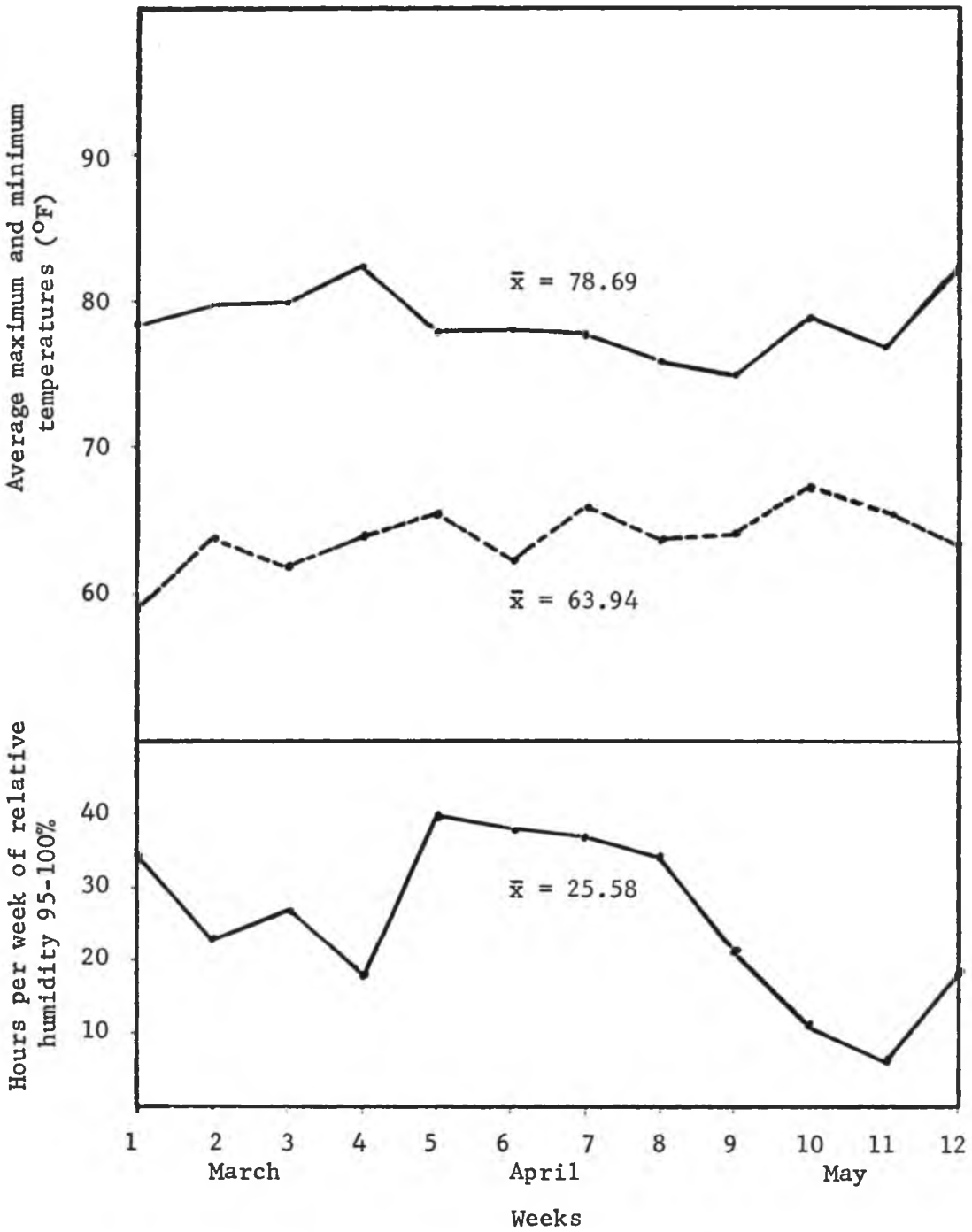


Figure 12. Weekly relative humidities in excess of 95%, and minimum and maximum temperatures in the corn field during March and May, 1974.

RESULTS

1. Average Rust Ratings

The mean rust ratings on a 1-7 scale of the 10 parents are given below, in order of increasing susceptibility:

Oh545	1.36	H95	4.79
CM105	1.73	C123	6.11
CM111	2.71	CM104	6.73
Mo17	3.37	AA8	6.80
CI21E	4.04	B37	6.90

Oh545 was the best resistance source, and CM105 was similarly high.

Average rust ratings of the 110 populations from these parents containing a total of 20,250 plants are summarized in Table 40. Average rust ratings of the 25 F_1 crosses was 4.34, about equal to the mid-point (4.46) between resistant parents (P_r) and susceptible (P_s). The range of the 25 F_1 hybrids was from 2.37 to 6.19.

Oh545 and CM105, possessing the highest resistance, clearly contributed high resistance in all crosses. All populations involving Oh545 and CM105 were consistently more resistant than those involving any other lines.

The average rust rating of the 25 F_2 populations was 4.53, slightly more susceptible than the F_1's, indicating some heterosis effect. F_2 averages of 17 populations, including four involving Oh545, deviated from F_1 values in the direction of greater susceptible. Eight populations, including four with CM111, deviated in the direction of greater resistance.

Summarized in appendix Table 1 are the statistics and frequency distributions of all progenies from the 25 matings, following their order in Table 40. The F_2 distributions regularly ranged to and

Table 40. Average rust ratings of parents, F_1, F_2, and backcross progenies

Cross (r X s)	Parent		Hybrid		Segr. generation		
	P_r	P_s	F_1	F_1-MP	F_2	B_r	B_s
Oh545 x AA8	1.36	6.80	3.31	-0.77	3.73	2.70	3.90
Oh545 x B37	1.36	6.90	3.03	-1.10	3.82	2.38	4.24
Oh545 x C123	1.36	6.11	2.86	-0.88	3.36	2.69	3.77
Oh545 x CM104	1.36	6.73	3.30	-0.75	2.78	2.26	3.48
Oh545 x H95	1.36	4.79	2.37	-0.71	2.78	2.26	3.48
CM105 x AA8	1.73	6.80	4.25	-0.02	4.50	3.50	5.30
CM105 x B37	1.73	6.90	3.49	-0.83	4.39	3.46	5.00
CM105 x C123	1.73	6.11	3.07	-0.85	3.97	3.29	3.72
CM105 x CM104	1.73	6.73	4.27	0.04	4.32	3.75	5.13
CM105 x H95	1.73	4.79	4.27	1.01	3.87	3.26	4.05
CM111 x AA8	2.71	6.80	5.11	0.35	4.73	4.34	5.39
CM111 x B37	2.71	6.90	4.40	-0.41	4.17	4.52	5.72
CM111 x C123	2.71	6.11	4.50	0.09	4.37	4.28	4.96
CM111 x CM104	2.71	6.73	4.81	0.09	4.21	3.99	5.75
CM111 x H95	2.71	4.79	3.39	-0.11	4.39	4.00	4.59
Mo17 x AA8	3.37	6.80	4.53	-0.56	4.95	4.59	5.45
Mo17 x B37	3.37	6.90	6.19	1.06	4.76	4.58	5.84
Mo17 x C123	3.37	6.11	4.87	0.13	4.70	4.44	5.44
Mo17 x CM104	3.37	6.73	4.86	-0.19	5.14	4.46	5.62
Mo17 x H95	3.37	4.79	4.09	0.01	4.31	3.87	4.88
CI21E x AA8	4.04	6.80	6.13	0.71	6.16	5.86	6.24
CI21E x B37	4.04	6.90	6.17	0.70	6.01	5.71	6.19
CI21E x C123	4.04	6.11	5.13	1.03	5.63	5.17	5.75
CI21E x CM104	4.04	6.73	5.33	-0.26	5.69	5.71	5.74
CI21E x H95	4.04	4.79	4.73	0.31	4.96	5.05	4.85
Average	2.64	6.27	4.33	0.14	4.53	4.01	5.01
No. plants	350	350	1729		7622	5155	5044

exceeded the parental means. In a few cases (e.g. CI21E X H95, Mo17 X H95) it would have been possible to select F_2 plants that were more resistant than the resistant parent.

The frequency distributions of the F_2 populations involving Oh545, CM105, and CM111 were somewhat bimodal, while those of other F_2 populations involving Mo17, were essentially normal about the F_1 means. Bimodality would seem to infer the action of major genes for resistance, segregating simply in these populations. The frequency distributions of the populations involving CI21E were skewed in the direction of the susceptible parental values.

Average rust ratings of B_r (backcross to resistant parent) and B_s (backcross to susceptible parent) were 4.01 and 5.01 respectively. The rust ratings of backcrosses were usually related to recurrent parental values, and the degree of association varied from cross to cross. Resistant backcrosses B_r averaged 4.01, exceeding the mid-point value (3.49) between resistant P_r (2.64) and the F_1 (4.33). However, susceptible backcrosses averaged 5.01, about equal to the mid-point(5.30) between susceptible P_s (6.27) and the F (4.33).

2. Components of Variance

Genetic variance: Genetic variance components of the 25 crosses are summarized in Table 41. The additive genetic variances averaged 55.1% of genetic variance for rust resistance. The non-additive genetic variance was of less importance, although much higher than estimated from diallel analyses.

Additive genetic variances for the 25 individual crosses varied greatly among the five resistance inbreds. Crosses involving the three most resistance inbreds, Oh545, CM105, and CM111 showed high additive

Table 41. Estimates of additive (V_A), non-additive (V_D), environmental (V_E) variances, narrow (nH) and broad (bH) sense heritabilities, and genetic advance (Gs) for rust resistance

Cross (r X s)	V_A	V_D	V_E	nH	bH	Gs
Oh545 x AA8	2.110	0.082	0.234	87.0	90.4	2.79
Oh545 x B37	2.140	-0.142	0.195	97.6	91.1	2.98
Oh545 x C123	1.206	0.443	0.269	62.9	86.0	1.79
Oh545 x CM104	1.622	0.097	0.254	82.2	87.1	2.38
Oh545 x H95	0.531	0.666	0.223	37.4	84.3	0.92
CM105 x AA8	0.945	0.989	0.306	42.2	86.4	1.30
CM105 x B37	1.471	0.801	0.240	58.6	90.4	1.91
CM105 x C123	1.110	0.531	0.348	55.8	82.5	1.62
CM105 x CM104	0.918	1.135	0.249	39.9	89.2	1.25
CM105 x H95	1.110	0.742	0.298	51.6	86.1	1.56
CM111 x AA8	0.836	1.108	0.273	37.7	87.7	1.16
CM111 x B37	0.727	1.394	0.229	30.9	90.3	0.95
CM111 x C123	0.909	1.024	0.352	39.8	84.6	1.24
CM111 x CM104	1.670	0.642	0.255	65.1	90.1	2.15
CM111 x H95	1.441	0.489	0.245	66.3	88.7	2.01
Mo17 x AA8	0.397	0.957	0.275	24.4	83.1	0.64
Mo17 x B37	0.577	0.888	0.218	34.3	87.0	0.92
Mo17 x C123	-0.521	1.385	0.296	-44.9	74.5	-1.00
Mo17 x CM104	0.547	0.895	0.264	32.1	84.5	0.86
Mo17 x H95	0.102	0.854	0.307	8.1	75.7	0.19
CI21E x AA8	0.032	0.532	0.280	3.8	66.9	0.07
CI21E x B37	0.114	0.534	0.306	11.9	67.9	0.24
CI21E x C123	0.142	0.644	0.337	12.6	70.0	0.28
CI21E x CM104	0.114	0.748	0.281	9.9	75.4	0.22
CI21E x H95	1.315	0.121	0.273	76.9	84.0	2.07
Average	0.863	0.702	0.272	47.0	83.4	1.22

genetic variances. These inbreds with high additive genetic variances appear to be most promising sources of rust resistance. Only one cross involving CI21E, CI21E X H95, showed high additive variance, possibly contributed by moderately susceptible inbred H95. These data and the frequency distribution of the individual plants in segregating generations suggest that there is no genes for rust resistance. A highly significant negative correlation (r=-0.63**) between F_1 disease ratings and their corresponding additive variances was calculated.

The non-additive or "dominance" genetic variance for the 25 individual crosses also varied substantially from cross to cross. In comparison with additive genetic variances, dominance in crosses involving Mo17 and CI21E made a major contribution to the inheritance of rust resistance (Table 41).

Environmental variance: Average environmental variance of the 25 crosses was 0.272 (Table 41), representing 14.8% of total phenotypic variance, ranging from 8.9% for Oh545 X B37 to 33.2% for CI21E X AA8. Environmental variances of the 25 individual crosses were consistent, and variations between crosses among the five resistant lines were small.

3. Heritability

Estimates of narrow sense heritability, based on the ratio of additive genetic variance to phenotypic variance, V_A/V_P, of the 25 individual crosses are summarized in Table 41. Average narrow sense heritability of the 25 crosses was 47.0 percent, ranging up to 97.6 percent for Oh545 X B37. Mo17 X C123 showed an unexpected negative heritability estimate. Narrow sense heritability estimates in crosses involving the three most resistant inbreds-Oh545, CM105, and CM111-were

always higher than those in crosses involving the moderate resistant inbreds, Mo17 and CI21E, reflecting the high additive genetic variance and low environmental variances in these crosses (Table 41).

Estimates of broad sense heritability, based on the ratio of total genotypic variance to phenotypic variance, V_G/V_P, of the 25 individual crosses are summarized in Table 41. Average broad sense heritability of the 25 crosses was very high, 83.4 percent. The range was from 66.9 for CI21E X AA8 to 91.1 percent for Oh545 X B37. Differences in broad sense heritability estimates among the 25 crosses were small in comparison to those of the narrow sense. Crosses involving CI21E showed relatively low broad sense heritability estimates.

4. Genetic Advances

Genetic advances through selection (Gs) were calculated by the equation proposed by Comstock and Robinson (1952):

$$Gs = i \cdot V_P \cdot H$$

where i represents the selection intensity, and is 2.06 at the 5 percent selection. V_P represents the phenotypic variance, and H represents the narrow sense heritability.

Genetic advances through selection in the 25 F_2 populations, assuming the top 5 percent of the resistant plants were selected, are summarized in Table 41. Average genetic advance per generation was 1.24 rust rating units, on the 7-point scale. Since the average rust rating of the 25 F_2 populations was 4.53 (Table 40), the 1.22 rust rating unit advance in one generation would amount to 26.9 percent increase for rust resistance.

5. Numbers of Gene Pairs

Minimum numbers of gene pairs controlling rust resistance were

calculated in two ways by the equations proposed by Weber (1950), and Castle and Wright (1921), respectively. The Weber formulas are slightly modified from the Castle-Wright formulas to correct for environmental variance.

The Castle-Wright formulas follows:

Minimum number of gene pairs,

$$\text{using } F_2 \text{ data} = \frac{D^2}{8\ (VF_2 - VF_1)}$$

$$\text{using } B_r \text{ or } B_s \text{ data} = \frac{D^2}{16\ (V_{B_r} \text{ or } _{B_s} - VF_1)}$$

where, D^2 = squared mean differences between two parents; and V represents variance.

Weber (1950) used a geometric average of the three non-segregating progenies for the estimates of environmental variances:

$\sqrt[3]{VP_r \times VP_s \times VF_1}$, instead of VF_1 in the Castle-Wright formulas.

Minimum numbers of gene pairs controlling resistance in the 25 F_2, 25 B_r, and 25 B_s by Weber and Castle-Wright formulas are summarized in Table 42. Castle-Wright formula could not be applied to two B_r populations involving CM105 because the F_1 variances were larger than B_r variances, respectively. When the F_2 data were used, average minimum numbers of gene pairs calculated from the 25 crosses by the Weber and Castle-Wright formulas were almost identical, accounting for 1.24 and 1.26, respectively. When the backcross data were used, Weber formula gave averages of 0.92 (B_r)and 0.94 (B_s) while Castle-Wright formula gave 0.83 (B_r) and 0.91 (B_s) gene pairs, respectively.

Table 42. Estimates of the minimum numbers of gene pairs controlling resistance to P. sorghi

	F_2		B_r		B_s	
Cross (r X s)	Weber*	C-W**	Weber	C-W	Weber	C-W
Oh545 x AA8	1.76	1.74	1.76	1.77	1.81	1.70
Oh545 x B37	2.00	1.98	2.59	2.55	1.99	1.97
Oh545 x C123	1.82	1.65	2.57	2.01	1.04	0.94
Oh545 x CM104	2.22	2.19	2.80	2.71	1.85	1.80
Oh545 x H95	1.34	1.24	1.24	1.08	0.69	0.64
CM105 x AA8	1.73	1.84	1.11	-- +	1.21	1.53
CM105 x B37	1.51	3.71	1.25	--	1.04	1.11
CM105 x C123	1.55	1.53	1.02	1.00	1.00	0.98
CM105 x CM104	1.60	1.55	0.97	0.93	1.14	1.09
CM105 x H95	0.66	0.68	0.45	0.46	0.53	0.55
CM111 x AA8	1.13	1.13	0.68	0.68	0.79	0.79
CM111 x B37	1.07	1.07	0.55	0.55	0.94	0.94
CM111 x C123	0.79	0.77	0.37	0.36	0.91	0.85
CM111 x CM104	0.91	0.88	0.65	0.61	0.85	0.79
CM111 x H95	0.30	0.28	0.20	0.18	0.32	0.28
Mo17 x AA8	1.16	1.12	0.66	0.62	0.74	0.70
Mo17 x B37	1.12	1.06	0.54	0.52	0.99	0.91
Mo17 x C123	1.21	0.95	0.37	0.32	0.58	0.46
Mo17 x CM104	1.05	0.96	0.52	0.48	0.89	1.05
Mo17 x H95	0.29	0.28	0.13	0.12	0.20	0.19
CI21E x AA8	2.00	1.34	0.82	0.56	1.42	0.90
CI21E x B37	1.70	1.97	0.73	0.82	1.32	1.68
CI21E x C123	0.78	0.62	0.34	0.28	0.62	0.44
CI21E x CM104	1.18	1.02	0.63	0.54	0.65	0.55
CI21E x H95	0.05	0.05	0.04	0.04	0.06	0.05
Average	1.24	1.26	0.92	0.83	0.94	0.91

*using Weber (1950) formula

**using Castle and Wright (1921) formula

+Formula not applicable

DISCUSSION

Generation mean analyses reveal that both additive and non-additive variances are important in the inheritance of rust resistance in maize. Additive genetic variance was a major contribution in the 25 crosses, supported by the results obtained from the 9 X 9 diallel analyses (Table 37). Crosses involving moderately resistant inbred, Mo17 and CI21E, showed high non-additive variances. However, the cross between moderately resistant CI21E and moderately susceptible H95, CI21E X H95, showed high additive genetic variance.

Gamble (1962) reported that additive genetic variance for grain yields in maize was increased when the experimental materials used were less selected in genetic background. In these aspects, CI21E and H95 might be interpreted as poorly selected for resistance. The importance of additive genetic variance in the inheritance of disease resistance in maize has been reported for Physoderma brown spot (Moll et al. 1963), Diplodia stalk rot (Russell 1961, Kappelman and Thompson 1966), corn stunt virus (Grogan and Rosenkranz 1968, Nelson and Scott 1973), northern corn leaf blight (Jenkins et al. 1954, Hughes and Hooker 1971), maize dwarf Mosaic virus (Josephson and Naidu 1971, Loesch and Zuber 1972).

High non-additive variances, detected in crosses involving Mo17 and CI21E, indicate that at least two major kinds of variances; additive and non-additive, are present for rust resistance in maize. These results are supported by the highly significant general and specific combining ability mean squares obtained in the 9 X 9 diallel analyses (Table 35). In the diallel analyses, it was recommended that both

general and specific combining ability effects are used to maximize genetic advance for rust resistance. Similar results have been reported in the resistance of maize dwarf mosaic virus (Josephson and Naidu 1971).

The frequency distributions of the F_2 individual plants (Appendix Table 1) showed, to some extent, complex genetic variances involved for rust resistance. The F_2 populations involving the three most resistant inbreds, Oh545, CM105, and CM111 showed bimodal distribution, those involving CI21E were skewed, and those involving Mo17 appeared normal. Normal distributions of the F_2 individual plants for rust resistance have been reported (Hooker 1967, Hooker 1969).

Two negative genetic variances were obtained in this experiment, the additive genetic variance for Mo17 X C123 and dominance variance for Oh545 x B37. The negative values can not be interpreted other than as sampling deviations, but may have values interpretable in the future (Dudley and Moll 1969). Estimates of negative genetic variances have been reported (Lindsey et al. 1962, Eunus 1964, Liang and Walter 1968, and Shin 1971). This discrepancy can occur due to invalidity of certain assumptions made in this experiment, e.g., that genes for rust resistance do not interact nor are linked.

The narrow sense heritability estimates from this study were somewhat less than those from diallel analysis. An average narrow sense heritability of 47 percent was estimated in this study, but 72 percent in the diallel analysis. Heritability estimates for the different populations often do not compare directly under different environmental conditions (Sprague 1966). The relatively low narrow sense heritability estimate in this study indicates that, to some extent, complex gene

actions are involved in the inheritance of rust resistance. However, high narrow sense heritability (averaged 82%) was obtained in crosses involving Oh545, except only one cross Oh545 X H95. The relativeness of Oh545 with H95 might result in the quite different heritability estimate.

The broad sense heritability estimates in this study, 83.4% from generation mean analysis and 85.8% from method 2 diallel analysis are similar to that of Hooker (1967), who obtained an average 85% broad sense heritability estimate from 45 F_2 populations.

The calculated minimum numbers of gene pairs controlling resistance revealed that relative small numbers of gene pairs are present in generalized rust resistance in maize. Castle (1921) proposed several assumptions for the accuracy of the estimated gene pairs. Among those assumptions, all individual rating scores must be distributed normally. The fact that rust ratings were not distributed normally (appendix Table 1) in several populations may precluded strict interpretation of the minimum gene pair data.

It should be possible to increase rust resistance by phenotypic recurrent selection or mass selection capitalizing on the additive genetic variance, without great difficulty. A breeding program based on highly additive genetic variances would be desirable for improvement of polygenic or generalized resistance to P. sorghi.

V. GENETIC POLYMORPHISMS OF RUST LESION LENGTH AND PUSTULE NUMBER

ABSTRACT

The inheritance of P. sorghi lesion length was studied in a 9 X 9 diallel. Highly significant variations for lesion length were found among genotypes. Heritability was high, accounting for 63% for broad sense and 57% for narrow sense. Additive genetic variance was a major portion of the total genotypic variance, accounting for 93%. Dominance variance was minor. Heterotic effect was extremely high, averaging 37%.

Inbred CI21E showed good generalized resistance in the field. However, it showed longer lesions than susceptible inbreds, Other data supported evidence that lesion length is inherited independently from pustule number. Highly susceptible inbred CM104 showed short lesions.

Highly significant general (GCA) and specific (SCA) combining ability mean squares were obtained. Moderate resistant inbred CI21E showed exceptionally high GCA effect for lesion length, while the best resistant inbred Oh545 showed exceptionally low GCA effects. It appears that resistant inbreds having long lesions should be excluded from selection for rust resistance in breeding programs.

The generalized resistance was studied at early vegetative stages in both field and greenhouse. Generalized resistance was accompanied by fewer pustules at early vegetative stages, suggesting that selection for generalized resistance prior to flowering under intense epiphytotics would be effective.

MATERIALS AND METHODS

Experimental materials used in this study were the 9 X 9 diallel crosses previously discussed in chapter 3. Rust lesion lengths were measured at three weeks after mid-silking from 10 plants per plot. Five clearly expressed lesions were selected on the leaf above the uppermost ear, and five lesions averaged. The 10 plant averages were obtained for four replications and were used for statistical analyses. Griffing's methods 2 and 4, model I and II (Griffing 1956) were employed for estimates of combining ability, and for variance and covariance components.

Ten inbred lines, comprising five with high generalized resistance and five susceptible lines were tested in a search for different number of pustules (immediately erupted spots) at the vegetative stage in the field and in the greenhouse conditions. The experiment was conducted in the field during July and October in 1973. A randomized complete block design with four replications was employed. Each plot consisted of 15 plants spaced 8 inches apart, with rows spaced 30 inches apart. Visible pustule were counted at 7 to 8 leaf stages from whole leaves on 10 individual plants per plot. Total pustule numbers per plot were used for statistical analysis. No artificial inoculation was made.

In the greenhouse, an experiment was conducted during April and May in 1974. Each inbred was planted in two 6-inch plastic pots, with five seedlings in nutrient-treated, sterilized soil. Corn plants were inoculated 30 days after planting with urediospore suspensions of *P. sorghi* collected from fresh corn leaves at the Waimanalo Farm.

Inoculum level was approximately 23,000 spores/1 ml. Inoculated plants were incubated in a plastic moisture chamber for sixteen hours and then moved to a greenhouse bench. Visible erupted pustules were counted from first and third leaves. Average number of pustules from two leaves of five plants per plot were used for statistical analyses. Bayes least significant difference (BLSD) (Duncan 1965) was employed for multiple comparisons for pustule numbers among genotypes.

RESULTS

1. Average Rust Lesion Length

Average rust lesion lengths of the nine parents and their 36 F_1 hybrids are summarized in Table 43. Parent, F_1, and grand means were 8.70, 11.52, and 10.96 mm, respectively. Considerable variations for lesion length were found among the parents and F_1 hybrids. The range of the parents was from 2.33 mm for Oh545 to 13.50 mm for B37, and that of the F_1 hybrids was from 6.18 for Oh545 X Mo17 to 19.28 for CI21E X B37. The parents B37, AA8, and CI21E had very long lesions (average = 12.94 mm), while the remaining parents had relative short lesions (average = 6.58 mm).

All F_1 hybrids showed longer lesions than their respective parents. Most hybrids involving CI21E showed exceptionally long lesions. The heterotic effects of the 36 F_1 hybrids were calculated as the ratios of F_1 hybrid to mid-parental values, and were extremely high, averaging 37.4% (Table 44).

2. Analyses of Variance and Covariance

Mean squares from the randomized block analyses of variance for parental and hybrid lesion length are summarized in Table 45, based on Griffing's methods 2 and 4. Highly significant differences were obtained among the 45 genotypes, following both methods of analysis (Table 45). General combining ability (GCA) and specific combining ability mean squares for lesion length were also highly significant (Table 46). Percent values obtained for the GCA and SCA variances were 51.1 and 47.4 for method 2 and 73.7 and 24.5 for method 4, respectively (Table 47). The latter may be viewed as more accurate, considering the high

Table 43. Average rust lesion length in mm of 9 parents and their 36 F_1 hybrids

Parent	CI21E	B37	AA8	Ant2D	Mo17	CM104	CM105	CM111	Oh545	Avg. of Hybrid
CI21E	11.93	19.28	18.13	19.23	14.25	15.73	12.95	13.55	11.15	15.53
B37		13.50	15.08	14.35	10.10	11.85	12.10	12.58	6.65	12.63
AA8			13.38	13.08	11.35	12.55	11.05	11.88	7.23	12.54
Ant2D				8.00	13.73	10.23	11.60	12.98	7.68	12.86
Mo17					9.33	10.13	13.15	8.63	6.18	10.94
CM104						8.68	7.90	8.65	7.33	10.55
CM105							5.98	7.48	8.00	10.53
CM111								5.13	7.13	10.36
Oh545									2.33	7.67

Grand mean = 10.96, Parental mean = 8.70, and F_1 mean = 11.52

se of treatment means based on parent and F_1 data = 0.388

se of the difference between two treatment means = 0.549

Table 44. Estimates of heterotic effects in percent for rust lesion length

Parent	B37	AA8	Ant2D	Mo17	CM104	CM105	CM111	Oh545
CI21E	12.20	43.26	92.98	34.05	52.64	44.61	58.85	56.38
B37		12.20	33.49	-11.52	6.85	24.23	35.05	-16.23
AA8			22.36	- 0.04	13.78	14.15	28.36	- 7.96
Ant2D				58.45	22.66	65.95	97.72	48.69
Mo17					12.49	71.78	19.36	6.00
CM104						7.78	25.71	33.15
CM105							34.65	92.54
CM111								91.15

Average heterotic effect in percent = 37.42

Table 45. Analyses of variance of rust lesion length in Table 43 based on Griffing's methods 2 and 4, fixed model I

Source	Method 2 d.f.	Method 2 Mean Square	Method 4 d.f.	Method 4 Mean Square
Genotypes	44	524.622**	35	476.643**
Replication	3	512.679	3	481.787
Genotypes x rep.	132	53.630	105	53.080
Error	1620	6.018	1296	6.509

**$P < 0.01$

Table 46. Combining ability mean squares based on methods 2 and 4, model I, for rust lesion length

Source	Method 2 d.f.	Method 2 Mean Square	Method 4 d.f.	Method 4 Mean Square
GCA	8	53.179**	8	44.487**
SCA	36	4.621**	27	2.266**
Error	1620	0.150	1296	0.163

**$P < 0.01$

Table 47. Variance components analyses for GCA and SCA value in Table 46

Components	Method 2 Variance	Method 2 Percent	Method 4 Variance	Method 4 Percent
GCA	4.821	51.06	6.332	73.65
SCA	4.470	47.35	2.103	24.46
Error	.151	1.59	.163	1.89

heterotic effect for lesion length.

It is perhaps more appropriate to interpret these lesion length data based on Random Model II, since the parents were chosen randomly with respect to lengths of lesions, and appear to broadly represent all maize germplasm. The GCA mean square based on method 4 was highly significant and SCA mean square was significant at the 5% level, based on a calculated error mean square of 1.327.

Estimates of phenotypic, genotypic, additive, dominance, and environmental variances based on random model II are summarized in Table 48. The genotypic variance was a major portion of the total phenotypic variance. Additive variance was a major portion of the genotypic variance, accounting for 92.8%. Non-additive genetic variance was negligible, accounting for 7.2% of the total genotypic variance.

Broad sense heritabilities based on both methods 2 and 4 were almost identical, accounting for 64 and 63% respectively. Narrow sense heritabilities were quite different from the results obtained methods 2 and 4, and accounted for 31 and 57%, respectively. Differences in narrow sense heritabilities were contributed by the high heterotic effects (37.4%), and the method 4 (F_1 only) in probably the most reliable.

3. General and Specific Combining Ability Effects

General combining ability effects for lesion length for susceptible parents were substantially higher than those for resistant (Table 49). The four parents CI21E, B37, AA8 and Ant2D showed positive GCA effects, indicating that these lines contributed to increase lesion length in hybrid combinations. The remaining six parents, however, showed negative GCA effects, indicating that these lines contributed to reduced

Table 48. Estimates of phenotypic variance and its components, and of broad (bH) and narrow (nH) sense heritabilities for rust lesion length

Variance	Method 2	Method 4
Phenotypic	18.127 ± 4.967	19.510 ± 6.395
Genotypic	12.109 ± 4.962	13.002 ± 6.390
Additive	8.829 ± 4.839	12.063 ± 6.358
Non-additive	3.280 ± 1.101	0.938 ± 0.643
Environmental	6.018 ± 0.212	6.509 ± 0.256
bH	64.1 (54.3 - 73.9)	62.6 (50.4 - 74.9)
nH	31.1 (3.0 - 59.2)	57.3 (43.5 - 71.1)

Table 49. Estimates of general combining ability effects based on method 4 for rust lesion length

Parent	Parental mean	Rust rating	GCA effects in mm
CI21E	11.93	4.15	4.58
B37	13.50	6.48	1.40
AA8	13.38	6.25	1.16
Ant2D	8.00	2.55	1.52
Mo17	9.33	2.98	-0.67
CM104	8.68	6.20	-1.12
CM105	5.98	2.30	-1.14
CM111	5.13	2.55	-1.33
Oh545	2.33	1.18	-4.41
se_d			0.216

lesion length. CI21E showed the highest GCA effects for lesion length, while Oh545 showed the lowest GCA effects. Line CI21E increased lesion length by 4.6 mm in crosses with other eight parental lines. In contrast, line Oh545 reduced lesion length by 4.4 mm in crosses with the other parental lines.

Specific combining ability effects for lesion length varied widely among the 36 hybrid combinations (Table 50). Mo17 X CM105 showed the highest SCA effect, 3.43, while B37 X Mo17 showed the lowest SCA effect, -2.15.

4. Pustule Numbers

Observations on five of the more resistant inbreds--Oh545, CM105, CM111, Mo17, and CI21E in order of increasing resistance--and five of the more susceptible--H95, C123, CM104, AA8, and B37 in order of increasing susceptibility--at the 7 to 8 leaf stage in both field and greenhouse conditions indicated that significant differences in numbers of rust pustules occurred (Table 51). In the field, the five lines shown to have generalized resistance at mature plant stage had fewer pustules at the early vegetative stage, likewise, the five susceptible lines had many more pustules. Average pustule numbers of the five resistant and five susceptible lines were 112 and 402 respectively. The ranges of the pustule number were from 41 to 191 in the resistant and from 257 to 407 in the susceptible lines. 5% Bayes LSD was 158.0 pustules. Although highly significant differences in pustule numbers among replications were also detected in the field, the mean square among genotypes showed highly significant difference atthe 1% level (Table 52). These results were supported by the results obtained in the greenhouse.

Table 50. Estimates of specific combining ability effects based on method 4 for rust lesion length in mm

Parent	CI21E	B37	AA8	Ant2D	Mo17	CM104	CM105	CM111	Oh545
CI21E									
B37	1.78								
AA8	0.86	0.99							
Ant2D	1.60	-0.09	-1.14						
Mo17	-1.19	-2.15	-0.67	1.35					
CM104	0.74	0.05	0.98	-1.70	0.39				
CM105	-2.02	0.32	-0.50	-0.31	3.43	-1.37			
CM111	-1.22	0.99	0.52	1.26	-0.90	-0.42	-1.58		
Oh545	-0.54	-1.88	-1.05	-0.96	-0.27	1.33	2.03	1.35	

se_d between effects of two crosses having one parent line in common:

Method 4 = 0.528

se_d between effects of two crosses having no parent line in common:

Method 4 = 0.482

Table 51. Average number of rust pustules from 10 inbreds

Inbred	Numbers of pustules In the field	In the Greenhouse
Oh545	89.0 a+	22.0 a
CM105	40.5 a	63.7 a
CM111	191.0 ab	64.9 a
Mo17	47.8 a	31.2 a
CI21E	189.0 ab	173.3 b
H95	443.5 d	320.2 c
C123	256.8 c	183.1 b
CM104	470.8 d	321.2 c
AA8	339.5 cd	246.0 bc
B37	497.0 d	178.8 b
5% BLSD	158.0	106.3

+Means within each group not having a letter in common are significantly different at the 5% level based on Bayes LSD Test.

Table 52. Analysis of variance of data in Table 51

Source	d.f.	Mean square Number of pustule In the field	d.f.	In the Greenhouse
Genotype	9	121216.30**	9	25317.46**
Replications	3	194836.29	1	0.29
Error	27	14798.16	9	3314.40
Total	39		19	

**P < 0.01

In the greenhouse, results were generally similar to those in the field. Average pustule numbers of the five resistant and five susceptible lines were 71 and 250 respectively. The ranges of the pustule numbers were from 22 to 173 in the resistant and from 179 to 321 in the susceptible lines, and the 5% LSD value was 106.3 pustules. B37 had relatively fewer pustules in the greenhouse than in the field. Mean square among plots was very small and did not show any significance statistically. However, mean square among genotypes was significant at the 1% level (Table 52).

DISCUSSION

Few studies have been made on the inheritance of lesion length in susceptibility to cereal diseases. The relationship between numbers of lesions and resistance has been reported for P. sorghi by Hooker (1973), for Helminthosporium maydis by Summer and Littrell (1974), and for rice blast caused by Pyricularia oryzae by Ou (1971).

Corn rust lesion length was highly heritable (63%) and significantly related to P. sorghi susceptibility. It is apparent that corn breeders should exclude long lesion plants in selection for rust resistance. Lim et al. (1974) reported that H. maydis lesion length at mid-silking was highly correlated with the susceptibility of cytoplasmic male sterile-T hybrids in maize ($r = 0.81$).

Resistant inbreds generally produced short lesions, but there were some exceptions. Moderate resistant inbred CI21E had quite long and aggressive lesions. In contrast, highly susceptible inbred CM104 had high numbers of relatively short lesions. The susceptibility of CM104 and the resistance of CI21E were emphasized by the results obtained in the genetic studies (Chap. 3 and 4).

Weak or moderate generalized resistant sources do not provide good breeding value for P. sorghi resistance. For example, crosses involving moderate resistant inbred CI21E always became more susceptible than the mid-parent value in the generation mean analyses. The frequency distributions of the individual F_2 plants in the populations involving CI21E were skewed toward susceptibility. The exceptionally high GCA effects of CI21E for lesion length contributed these causes.

Highly resistant inbred Oh545 showed exceptionally short lesions

with high negative GCA effect. The outstanding resistance of Oh545 was evidenced from the results obtained in four seasonal tests of the 40 inbreds, diallel, and generation mean analyses.

An extremely high heterotic effect (37.4%) obtained in this study revealed that hybrid vigor contributes increased susceptibility in maize under severe P. sorghi epiphytotics. However, it has been reported that most of the widely used inbreds and hybrids have good level of generalized resistance to P. sorghi as it occurs in temperate regions (Ullstrup 1955, Russell and Hooker 1959, Hooker 1962).

VI. PEROXIDASE ACTIVITY ASSOCIATED WITH *PUCCINIA SORGHI* INFECTION IN MAIZE

ABSTRACT

Five parents and their 10 F_1 crosses of maize (Zea mays L.) were tested by means of diallel analysis for the inheritance of peroxidase activity following *Puccinia sorghi* rust infection. Peroxidase activity was measured at day zero and at 2 days and 8 days after inoculation. Peroxidase activity was increased significantly by *P. sorghi* infection in all 15 genotypes after 8 days, but not after 2 days.

Highly significant differences in peroxidase activity were detected among the 15 genotypes. The highly generalized resistant inbred, CM105, and its hybrids showed exceptionally high peroxidase activity in both healthy and infected plants. However, another highly resistant inbred, Oh545 showed exceptionally low peroxidase activity.

Significant general combining ability and specific combining ability mean squares were detected for peroxidase activity independent of disease. GCA mean squares, however, were consistently a major contribution to the inheritance of peroxidase activity in the infected plants whereas SCA mean squares were a minor contribution.

Rust resistant maize plants controlling monogenic dominant $Rp_1{}^d$ gene showed stronger peroxidase responses than their susceptible counterparts in the gel electrophoresis and densiometric tracings. The increased peroxidase activity occurred in both major leaf peroxidases, Px_3 and Px_7. It was especially reflected in the appearance or great increase of activity in 'daughter bands' or metazymes of the Px_3 locus more rapid in migration than the original bands.

MATERIALS AND METHODS

Plant materials (5 X 5 diallel) and culture : Five maize inbreds were selected for this study, including two generalized rust resistant lines, Oh545 and CM105 and three highly susceptible lines, AA8, B37, and CM104. All possible crosses, excluding reciprocals, were made in 1972. The five parents and 10 F_1 hybrids were grown in the greenhouse conditions in August 1973. Each entry consisting of five seedlings in 3 replicated 6-inch plastic pots containing a sterilized soil with nutrients.

Rust inoculation : Inoculation was made two weeks after planting when the seedlings were in the 4 to 5 leaf stage with the second leaf fully expanded. All experimental plants were sprayed with *P. sorghi* urediospore suspensions, approximately 30,000 spores/ml. The urediospores were collected from fresh corn leaf tissues at the Waimanalo Farm. Control plants were sprayed with water. All plants were incubated in moist chambers consisting of inverted plastic garbage cans for 24 hours and then moved to a greenhouse bench.

Enzyme preparation and assay : Six same leaves per entry were taken immediately before inoculation, and at two and eight days after inoculation, respectively. One disc (1 cm diameter) was taken from each of the six leaves, and two disc samples combined to provide three samples for peroxidase assay. Samples were ground in 5 ml phosphate buffer at pH 6.0. The macerated suspensions were centrifuged at 10,000 rpm for 10 min and the supernatant was used as an enzyme source. Preparations such as these include largely the cytoplasmic fraction; the wall-bound fraction of young leaves, estimated to represent 30% of the total peroxidase activity, was not included.

Peroxidase activity was measured on a Bausch & Lomb colorimeter at room temperature. The change in absorbancy at 470 nm of guaiaco substrate in the presence of hydrogen peroxide and enzyme was recorded for 3 min. The guaiacol test solution consisted of 5.4×10^{-4} ml guaiacol and 3.5×10^{-5} ml hydrogen peroxide in 0.02 M phosphate buffer (pH 6.0) made up to 3.0 ml final volume. The Lowry method (Lowry et al. 1951) was used for protein determinations, with crystalline bovine serum albumin as the standard. Peroxidase activity was expressed as enzyme activity in O. D. unit/min./protein content (mg/ml), and analyzed by Griffing's method 2, model I (Griffing 1956) to estimate general and specific combining ability.

Electrophoretic procedures followed methods described by Brewbaker et al. (1968). The standard horizontal gels were of 7% polyacrylamide and were electrophoresced using lithium-borate buffer at pH 8.1 for approximately 10 hours at 8 v/cm and at 4^{o}C. Benzidine dihydrochloride and o-dianisidine were used for staining gels, and stained gels were rinsed in running water.

Densitometric analyses were carried out after staining the electrophoresced gels with o-dianisidine and rinsing with 50% ethanol and running water. The gels was transferred to a Model 542 Densicord to trace the peroxidase bands using a 0.1 X 6 nm slit aperture and a 465 nm filter.

RESULTS

Peroxidase activity : Average peroxidase activities of 15 genotypes involving 5 maize inbred lines and their hybrids are summarized in Table 53. Data were taken prior to inoculation, and at 2 and 8 days thereafter. Wide differences were found among the 15 genotypes for average peroxidase activity, ranging from 1.06 for Oh545 to 4.22 for CM105. Inbred CM105 and all crosses involving CM105 had greater than average peroxidase activity (2.07). Peroxidase activities of CM105 were almost always the highest among the 15 genotypes, although its hybrids were commonly low in activity. Two U. S. Corn Belt inbreds, B37 (rust susc.) and Oh545 (res.) showed relatively low peroxidase activities.

The average peroxidase activities of the 5 genotypes at 8 days following inoculation were greater than control (2.66 vs 1.96). Inoculated leaves exceeded controls in every one of the 15 genotypes, despite wide differences among controls.

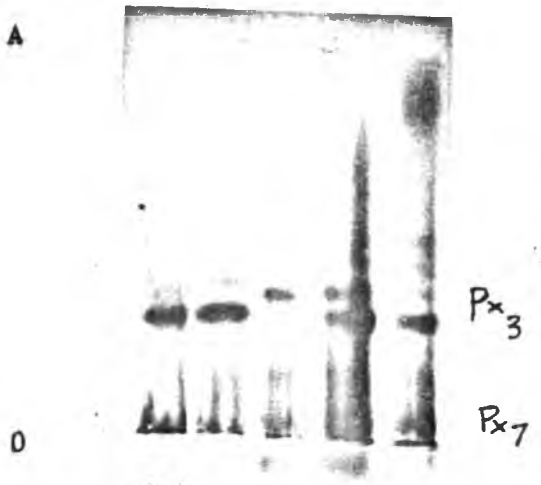
Zymograms of peroxidases from selected treatments are shown in Figure 13 through 16. Two major enzymes, Px_3 and Px_7 dominate the leaf samples used in this study. Little change in zymograms occurred at 2 days following inoculation (Fig. 14), but inoculated samples showed evident increase in both Px_3 and Px_7 after 8 days (Fig. 15, 16). The major evidence of increase is in the "daughter bands" or metazymes generated from the Px_3 band and of increasing mobility on gels, best seen for inoculated AA8 X CM104 in Fig. 16.

Analyses of variances : Mean squares from the randomized block analyses of variances of the 15 genotypes for the peroxidase activity are summarized in Table 54. Highly significant differences were detected

Table 53. Average peroxidase activity for 5 parents and their 10 F_1 hybrids

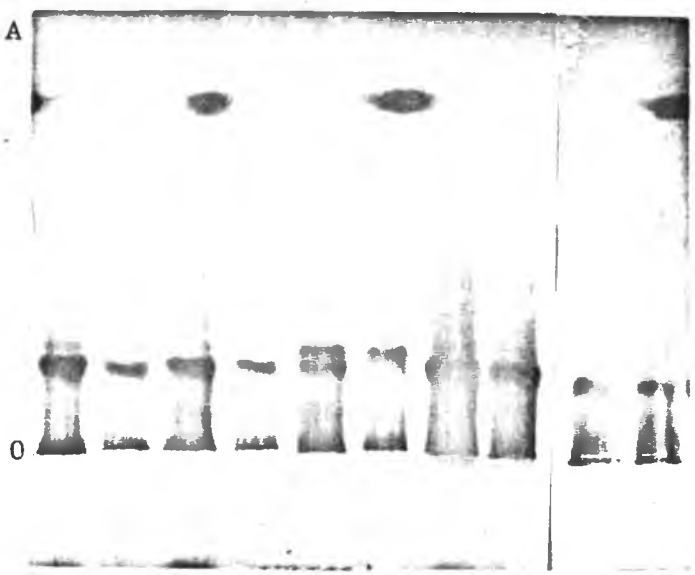
Entry	Peroxidase activity in A/min/mg protein					
	Day 0	Day 2		Day 8		
	C+	C	I	C	I	Average
AA8	1.78	2.47	2.12	1.92	3.19	2.30
B37	0.73	1.56	1.26	1.29	1.34	1.24
CM104	1.63	3.42	3.02	1.53	2.38	2.40
CM105	2.51	2.66	3.07	6.33	6.54	4.22
Oh545	0.78	0.80	1.01	1.21	1.50	1.06
AA8 x B37	0.85	1.64	1.49	1.47	2.26	1.54
AA8 x CM104	1.38	2.11	2.10	1.39	2.08	1.81
AA8 x CM105	2.42	2.21	2.85	2.92	3.32	2.74
AA8 x Oh545	1.38	1.47	1.30	1.12	2.36	1.53
B37 x CM104	1.46	1.31	1.71	1.40	1.89	1.55
B37 x CM105	2.30	2.37	2.50	1.52	1.81	2.10
B37 x Oh545	0.94	2.43	1.06	1.10	2.52	1.61
CM104 x CM105	2.61	3.37	2.85	2.49	4.15	3.09
CM104 x Oh545	1.30	1.88	1.87	1.96	2.09	1.82
CM105 x Oh545	1.47	1.98	2.32	1.76	3.53	2.21
Avg.	1.57	2.11	2.03	1.96	2.66	2.07
se of treat. mean	0.24	0.32	0.33	0.23	0.41	0.31
se_d Between two treat. mean	0.35	0.46	0.47	0.32	0.57	0.43

+C = Control, I = Inoculated.



AA8 B37 CM CM105 Oh545

Fig. 1. Anodal peroxidases of five maize inbreds.



C I C I C I C I C I

AA8 B37 CM104 CM105 Oh545

Fig. 2. Peroxidases of five maize inbreds at 2 days after inoculation by P. sorghi (D_2). C samples were controls, I samples were infected.

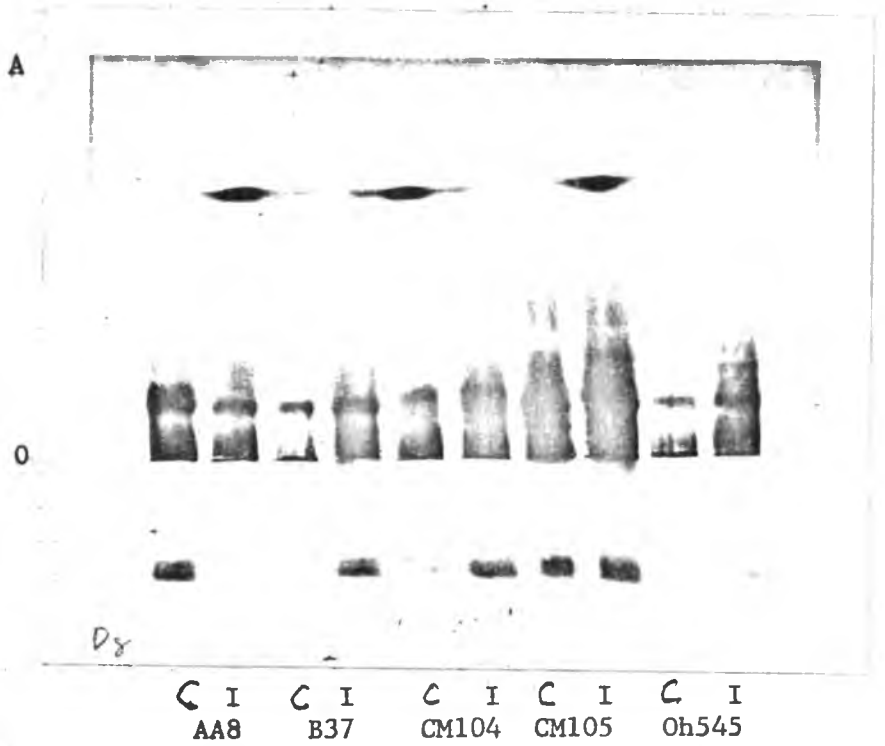


Fig. 13. Peroxidase ... of maize inbreds at 8 days after inoculation (D_8). C samples were controls, I samples were infected.

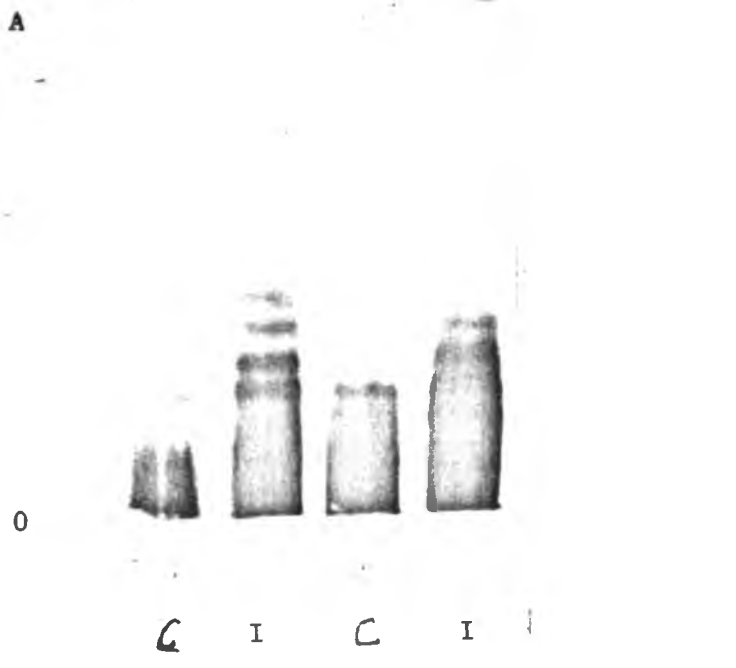


Fig. 14. Peroxidases response of two F_1 crosses at 8 days after inoculation (D_8). C samples were controls, I samples were infected.

Table 54. Mean squares from randomized block analyses of variance for peroxidase activity

		Mean squares					
		Day 0	Day 2		Day 8		
Source	d.f.	C	C	I	C	I	Average
Genotype	14	1.21**	1.56**	1.55**	5.16**	3.73**	2.88**
Replication	2	0.34	2.52	2.82	0.79	0.17	1.33
Error	28	0.19	0.31	0.34	0.16	0.49	0.30

Table 55. Combining ability mean squares for peroxidase activity

		Mean squares					
		Day 0	Day 2		Day 8		
Source	d.f.	C	C	I	C	I	Average
GCA	4	1.18**	1.04**	1.68**	4.30**	3.32**	2.30**
SCA	10	0.09	0.31*	0.05	0.69**	0.41*	0.31*
Error	28	0.06	0.10	0.11	0.05	0.16	0.10

Table 56. Variance components[+] in percent for combining ability based on data in Table 55.

	Variance components in percent					
	Day 0	Day 2		Day 8		
Source	C	C	I	C	I	Average
GCA	63.2	29.8	81.8	46.8	52.4	54.8
SCA	13.3	46.8	-22.7	49.1	28.6	23.0
Error	23.5	23.4	40.9	4.04	19.1	22.2

[+]GCA = (GCA MS - Error MS) / (No. of parents + 2)

SCA = SCA MS - Error MS

among the 15 genotypes at all dates.

General (GCA) and specific combining ability (SCA) mean squares of the 15 genotypes for the peroxidase activity are summarized in Table 55, and the variance components converted to percents in Table 56. General combining ability mean squares were all highly significant, expressed also by a GCA value of 54.8 percent.

Specific combining ability mean squares were generally small, although the average was significant at the 5% level. The minor importance of the SCA is also expressed by the 22.2 per cent average SCA variance component (Table 56).

Estimates of the general combining ability effects of the five parents for peroxidase activity are summarized in Table 57. Wide differences of the GCA effects among the five parents were detected. The GCA effects of the parents ranged from -0.45 for both B37 and Oh545 to 0.83 for CM105. GCA effects for CM105 were generally the greatest in the individual treatments, except those for day 2 control plants. SCA effects for 10 crosses varied substantially (Fig. 58).

Peroxidase reactions of monogenic genotypes : Seven paired double-cross hybrids, genetically similar except for gene, Rp_1^d, conferring complete resistance to P. sorghi were also tested for peroxidase activity. These hybrids were grown in paired rows under severe natural rust epiphytotics prevailing at the Waimanalo Farm during April and August, 1973. Disc samples (see Fig. 17) of 2 cm diameter were taken from resistant and susceptible plants at flowering time. All resistant plants were also heavely infected (Fig. 17). Electrophoretic peroxidase responses of seven paired double-cross hybrid leaves to infection by P. sorghi are presented in Fig. 18. Leaf samples of plants with the Rp gene had strong

Table 57. Estimates of general combining ability effects for peroxidase activity

Parent	General combining ability effects					
	Day 0	Day 2		Day 8		
	C	C	I	C	I	Average
AA8	0.02	-0.04	-0.03	-0.15	0.06	-0.03
B37	-0.34	-0.26	-0.42	-0.52	-0.69	-0.45
CM104	0.09	0.41	0.34	-0.21	-0.14	0.10
Oh545	-0.40	-0.47	-0.52	-0.49	-0.35	-0.45
se_d	0.13	0.17	0.18	0.12	0.22	0.16

Table 58. Estimates of specific combining ability effects for peroxidase activity

Cross	Specific combining ability effects					
	Day 0	Day 2		Day 8		
	C	C	I	C	I	Average
AA8 x B37	0.40	-0.17	-0.09	0.19	0.22	-0.05
AA8 x CM104	-0.30	-0.37	-0.24	-0.21	-0.50	-0.32
AA8 x CM105	0.19	-0.23	0.21	-0.26	-0.53	-0.22
AA8 x Oh545	0.18	-0.13	-0.18	-0.21	-0.01	-0.07
B37 x CM104	0.15	-0.95	-0.24	0.17	0.06	-0.16
B37 x CM105	0.45	0.15	0.25	-1.28	-1.30	-0.35
B37 x Oh545	0.11	1.05	-0.03	0.15	0.90	0.44
CM104 x CM105	0.33	0.48	-0.16	-0.63	0.50	0.10
CM104 x Oh545	0.04	-0.17	0.13	0.70	-0.17	0.11
CM105 x Oh545	-0.33	-0.02	0.17	-1.09	0.09	-0.24
Avg.	0.04	-0.04	-0.02	-0.25	-0.07	0.0
se_d between effects of two parent lines	0.23	0.30	0.31	0.21	0.38	0.29
se_d between 2 crosses having one parent line in common	0.32	0.42	0.44	0.30	0.53	0.40
se_d between 2 crosses having no parents in common	0.29	0.39	0.40	0.27	0.48	0.37

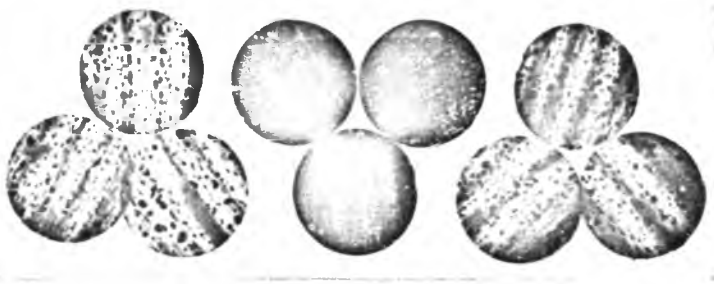
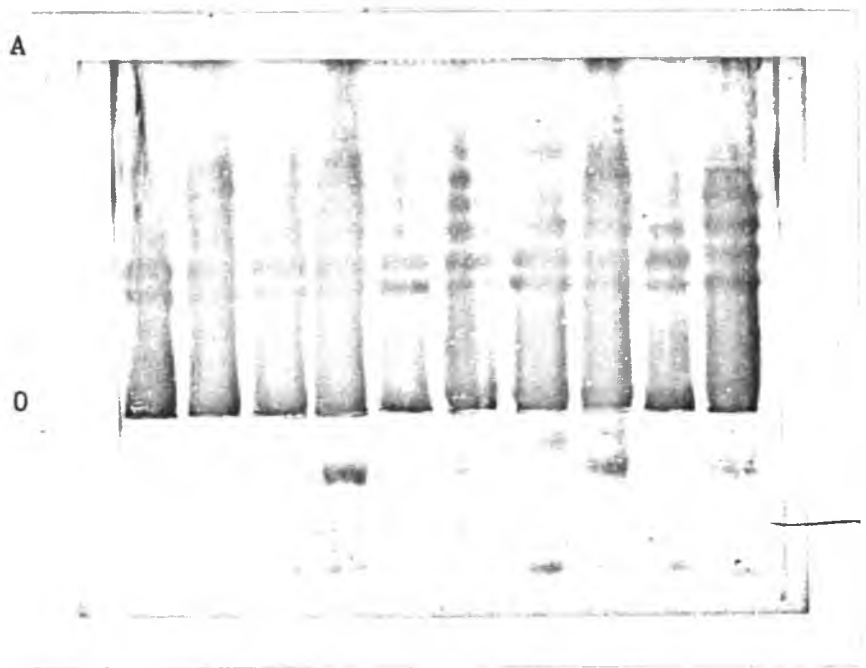


Fig. 15. Disk samples of rust infected leaves from susceptible (left, right) and resistant (center) genotypes.



include 681 too!

Fig. 16. Peroxidase response of five double-cross hybrid leaves to infection by P. sorghi; rp samples were susceptible, Rp samples were resistant.

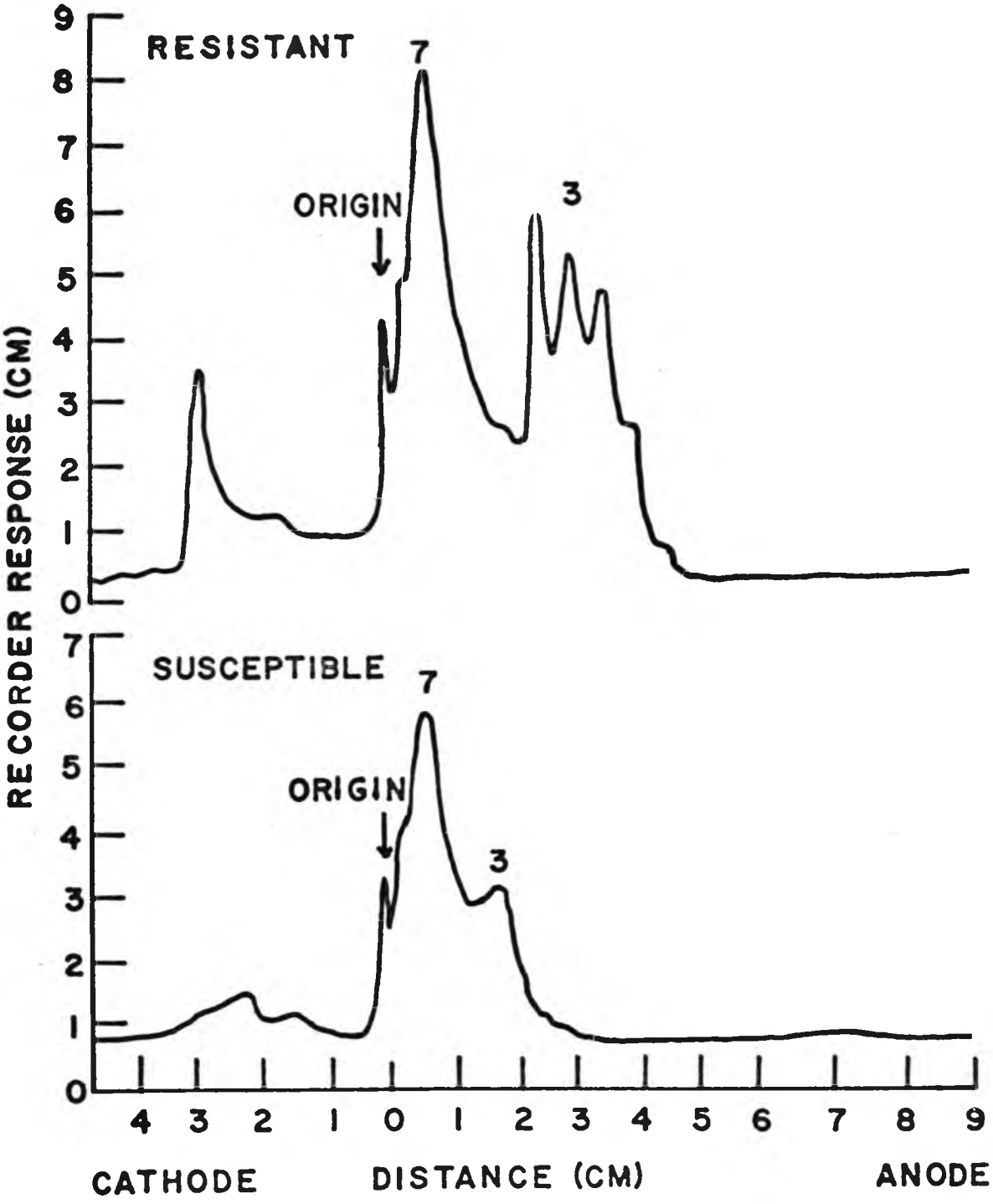


Figure 19. Densitometric tracings of peroxidase activity of resistant and susceptible hybrid 681.

peroxidase responses in both Px_3 and Px_7 in all trials, staining with benzidine dihydrochloride and o-dianisidine (Fig. 18). Rust infected Rp samples had several more daughter bands than their susceptible counterparts.

Densitometric tracings of peroxidase activity of one of the paired hybrid gels are presented in Fig. 19. Resistant hybrid 681 had higher peroxidase activity and had at least two more daughter bands in Px_3 than its susceptible counterpart. Peroxidase activity of Px_7 in the resistant hybrid also seemed to be higher than its susceptible counterpart.

DISCUSSION

Quantitative increases in peroxidase activity were significant 8 days after P. sorghi rust infection in maize, but not after 2 days. Increases of peroxidase activity after pathogen infection have been reported in maize with Helminthosporium carbonum infection by Jennings et al. (1969), in tomato with Phytophthora infection by Pierce and Brewbaker (1973), in sweet potato with Colletotrichum destructivum by Yu and Hampton (1964), in flax with Melampsora lini by Andreev and Shaw (1965), and in beans with virus by Farkas and Stahmann (1966).

The increase of peroxidase enzymes following P. sorghi infection in maize principally occurred in two major enzymes, Px_3 and Px_7, with somewhat greater increase of Px_3. The Px_3 and Px_7 enzymes dominate the leaf samples: Px_7, immediately anodal of the origin and somewhat diffused, and the anodal Px_3 enzymes at about Rf 30 in zymograms (Brewbaker and Hasegawa 1974). Several metazymes or daughter bands appeared in Px_3 after 8 days infection with P. sorghi.

Amounts of peroxidase activity in healthy and infected maize leaves were not directly related to generalized rust resistance, since one highly resistant inbred CM105 showed exceptionally low peroxidase activity. Jennings et al. (1969) reported that a plant resistant to H. carbonum in maize showed higher peroxidase activity than its healthy counterpart, and varied substantially in leaves infected with different pathogen races.

General combining ability variances were a major contribution to the inheritance of peroxidase activity following P. sorghi infection, but specific combining ability variances were a major contribution in healthy plants. No previous report has been made combining ability estimates

in peroxidase activity. Levings et al. (1971) reported that gene action of an auxin-inducible peroxidase in oat was dominant based on monohybrid ratio to the null condition. Genetic control of plant isozymes has been demonstrated by Scandalios (1969), MacDonald and Brewbaker (1972), and Pierce and Brewbaker (1973). Pierce and Brewbaker emphasized that genetic control of isozyme polymorphisms appeared to be largely monogenic, with rareoligogenic control.

Monogenic rust resistant plants controlled by dominant gene, Rp_1^d, showed stronger peroxidase responses than their susceptible counterparts. The increase of peroxidase activity mostly occurred in Px_3 and Px_7. Brewbaker and Hasegawa (1974) described twelve major peroxidases in maize. Among these 12 peroxidases, Px_3 and Px_7 appeared almost exclusively in leaf tissues. Seevers et al. (1971) studied the role of peroxidases in rust resistance in wheat and detected 14 peroxidase isozymes in both healthy and infected leaves. Only 1 (isozyme 9) of the 14 isozymes was associated consistently with the development of rust resistance at 20°C. Additional evidence was obtained that the isozyme 9 accounted for the increased peroxidase activity observed in extracts from resistant plants. Daly (1971) reviewed thoroughly these aspects of the rust resistance in wheat.

VII. INHERITANCE OF HUSK NUMBER IN MAIZE

ABSTRACT

A preliminary study revealed that husk number in a maize composite was significantly correlated with earworm damage ($r = -.396$). Forty-three widely used inbred lines were studied for husk number, leaf number, and days to mid-silk. Most of the lines with high husk number were late inbreds with high leaf numbers originating from the tropics or southern temperate regions. Husk numbers were significantly correlated with leaf number at the 1% level ($r = .549$) and with days to mid-silk at the 5% level ($r = .384$).

Among the 37 maize races tested, five races averaged more than 15 husks and two races had fewer than 7 husks. Zapalote Grande had the highest husk number, 19, while Sabanero blanco had only 6 husks. Large variations were observed among the 37 maize races for husk number.

Combining ability estimates for husk number were obtained using a 9 X 9 diallel. Considerable variations in husk number among the 45 F_1 genotypes were found. CM111 and CM105 had high husk numbers with high GCA (general combining ability) effects. Highly significant GCA and SCA (specific combining ability) mean squares were detected, with GCA mean squares always larger than SCA. Broad and narrow sense heritability estimates were 61% and 51%, respectively. There was a +11.2% heterotic effect in F_1 hybrids over their mid-parent values. A highly significant coefficient of linear determination ($r^2 = .769$) was calculated between parental husk number and their respective GCA effects. It should be possible to increase husk number and improve earworm resistance by various breeding methods such as recurrent selection, capitalizing on additive genetic variance.

LITERATURE REVIEW

No plant-feeding insect equals the corn earworm, Heliothis zea Boddie, in its total destructiveness each year, when one considers its many hosts throughout the world (Painter 1968). Annual corn yield losses due to corn earworm were estimated as 4.0% for the period of 1951 to 1960, approximately equal to 160 million dollars based on 4 billion bushels of corn (Anonymous 1965).

Little is known about the inheritance of corn husk numbers, which may be of significance in the resistance to earworm in maize. However, other husk characters have been studied extensively in relation to earwork resistance (Collins and Kempton 1917; Phillips and King 1923; McClelland 1929; Phillips and Barber 1931; Hawthorn and Fletcher 1934; Blackwell 1930; Poole 1935, 1937, and 1940; Douglas 1947; Walter 1961; Valle and Miller; Painter 1968; Widstrom et al. 1970; and Wiseman et al. 1970). The following review covers pertinent papers in chronological order.

Collins and Kempton (1917) did the most extensive and earliest work to study corn earworm resistance, and proposed hybridization as a method to reduce earworm damage. Husk number was one of the four characters considered to protect against earworm damage at the beginning of their experiment. The other three characters were husk extension beyond the tip of the ear, thickness of the husk covering, and the texture of the husks. They measured amount of damage, number of larvae, and damage per larva. All three traits showed significant negative correlations with number of husk layers, husk extension, and husk length. But husk number showed poor negative non-significant correlations with damage (-.08),

with number of larvae (-.15), and with damage per larva (-.01). A significant positive correlation between silking date and earworm damage was obtained, recently conferred by Widstrom and Starks (1967). However, Yarnell (1952) found no relationship between the silking date and the earworm damage.

McClelland (1929) and Poole (1937) observed little relationship between husk length and earworm resistance both in sweet corn and field corn. Poole (1935) and Walker and Anderson (1938) believed that husk extension beyond the tip of the ear was not directly associated with the earworm resistance, except as accompanied by tightness of tip cover. Poole observed a significant correlation in six out of 39 varieties. Recently, Starks and McMillion (1967) confirmed no correlation between these traits in their materials.

Poole (1935, 1936) suggested that the principal factors conferring earworm resistance were very likely the tightness and number of husks, or volatile chemical compounds. Poole (1940) reported that southern corn varieties were generally high in husk number. The highest type of resistance was associated with southern dent corns that were well adapted in tropical or subtropical regions where earworms thrive throughout the year from pre-Colombian times.

In the north, where the earworm does not thrive and early maturity is necessary, earworm damage has been considered of minor economic importance. By contrast, in the south Del Valle and Miller (1963) indicated that heavy and tight husks were needed for earworm protection. In addition to this, it was desirable to have ears well covered with a tight, thick husk to prevent damage caused by the water penetration and the development of fungi and other micro-organisms. This could also

reduce the damage due to birds.

Phillips and Barker (1931) studied the value of husk protection provided by tightness and length of husk. Generally, varieties with long tight husks were more resistant to earworms than those with short loose husks. They concluded that the most effective protection was provided by five-inch husk extension beyond the tip of the ear that was tightly wrapped throughout its entire husk length. This type of husk cover also provided a long narrow channel limiting the activity of the earworm inside the husks.

Burk et al. (1936) observed a 17.4% increase of marketable ears as results of tightness of husk, and found no evidence that husk length influenced the amount of earworm damage. Blanchard et al. (1941) reported that inbreds differed greatly in the amount of damage, and that the resistance was heritable.

Painter and Brunson (1940) suggested that selection in open-pollinated varieties would make possible the discovery of higher degrees of earworm resistance. Dicke and Jenkins (1945) observed differences in susceptibility to corn earworm damage in fieldcorn hybrid. They reported that tight husks, extending at least an inch beyond the tip and flinty grains with hard texture near the tip of the ear offered the best protection against earworm damage. Long-husked inbreds including 317Lh showed outstanding earworm resistance. Douglas (1947) found that a 2- or 3- inch husk extension beyond the tip conferred as much protection on field corn from earworm damage as a 5- or 6- inch extension.

For manual infestation of earworm of maize plants, Blanchard and Satterthwait (1942) found that from five to seven larvae per ear, two to four placed at one time and the remainder after 3 to 6 days, resulted

in the heaviest infestations of test ears.

Luckmann et al. (1964) reported that silk balling appeared to provide a physical barrier to earworm penetration expressed by mass or pressure or both. In corn lines having tight husks the earworm may feed to the silk ball and chew out through the husk or stop before reaching the ball. They was concluded that these kinds of reactions due to silk balling was associated with resistance and might be the only form of resistance in some lines.

The influence of several climatic conditions on the degree of earworm damage has been reported (Widstrom and Starks 1967, Widstrom et al. 1970). Widstrom et al. (1970) has conducted extensive studies on the climatic conditions and plant characteristics (14 characters measured) related to earworm resistance in maize. They reported that husk characters gave reliable information to identify resistant plant genotypes from the earworm damage. They concluded that both physical and chemical factors conferred resistance to earworm damage in maize. The mechanical protection of tight husks reduced earworm damage substantially. Correlation values (r) of corn earworm damage with husk protection and with husk tightness were significant, -.47 and -.33, respectively. The following criteria were used to estimate injury, husk tightness, and husk protection:

Corn earworm injury: 0=no damage, 1=silk damage only, 2=damage to 1 cm depth below the ear tip, and 3 ..... n = 1 added for each additional centimeter of damage (Revised Scale).

Husk tightness: Visual rating of 0 = very loose with ear exposed through 5 = extremely tight.

Husk protection: The value for corn earworm injury when husks were slit, corrected for corn earworm injury based on the Revised

Scale in the same line.

Painter (1968) summarized the chief factors affecting damage in maize by the earworm as follows: (1) Value as food for larvae (2) Attractiveness for oviposition (3) Hardness of the kernels (4) Tightness of husks (5) Husk extension (6) Number of husk leaves (7) Number of husk layers etc.'

Most studies on the inheritance of earworm resistance have been done by Widstrom and his colleagues at Tifton, Georgia. Widstrom and Davis (1967) found significant levels of general and specific combining ability for corn earworm damage in two diallel sets involving five and six sweet corn inbreds. Crosses between the two most resistant inbreds showed the most resistance, but crosses between the two most susceptible inbreds did not show the most susceptibility. Reciprocal and maternal effects in F_1 hybrids were detected.

Widstrom and Hamm (1969) also estimated highly significant general combining ability effects for corn earworm resistance, while specific combining ability effects were low. Highly significant genotype X environment interactions were attributed to the interaction of general combining ability effects with year X location. They recommended utilizing both general and specific combining ability effects for better potential selection progress.

Widstrom et al. (1970) estimated reduction of earworm damage for several cycles of selection progress based on recurrent selection. The selection progress for utilizing specific combining ability reached a plateau after approximately three or four generations of half-sib selection. They suggested that the use of highly susceptible testers for identification of the better resistant progenies. It was also

demonstrated that the selection progress for earworm resistance could be developed simultaneously with selection for desirable agronomic characteristics on the same plants.

Zuber et al. (1971) reduced earworm damage by an average of 2.8% each generation by mass selection in two synthetic varieties for 10 generations during the period 1956 to 1965. They concluded that the mass selection in synthetic populations for earworm resistance was effective in maize.

Widstrom et al. (1972) estimated genetic variances for the earworm damage for three composite varieties derived from Latin American sources. Additive genetic variances in these three populations were much higher than dominance variances. They observed high heritability and genetic advances in selection. In general, heterosis was not significant in the hybrid combination. However, dominance played a significant role in selection for resistant plant types. Widstrom and McMillian (1973) found that additive, dominance, and epistatic variances were all significant for earworm resistance in sweet and dent corn crosses tested.

Wiseman et al. (1970) found that some selected top ranking performanced southern hybrids had husk and / or kernel resistant to earworm damage. The husks of the selected hybrids provided a significant protection against the ravages of several insect species including earworm and contributed substantially to increased yield.

Straub et al. (1973) found that resistant lines or hybrids had less pupal productivity. Among the strains tested, (Mo17 X Mo5) X Zapalote Chico had the largest pupae, while resistant inbred Ab18 had the smallest pupae. They believed that chemical silk resistance was partially manifest in the influence upon larval growth rate and the ensuing delay in pupation. Resistant maize race Zapalote Chico had

only 20% larval recovery of the average of 53.1% and was indicated as an antibiosis factor.

Several heritable, morphological characters conferring resistance to corn earworm have been reviewed. It is notoriously difficult to predict which lines or varieties will transmit high earworm resistance to others; establishment of criteria assisting the plant breeder in selection for resistance combining ability would be of great value.

Heliothis zea (Boddie) was found as the princial insect pest on the ears of sweet corn in wouth Florida, being particularly abundant in spring when control is most difficult (Janes 1973).

The corn earworm is also abundant and ubiquitous in Hawaii, and a major deterrent to sweet corn production in year-round culture. Losses are estimated at over 35% of the kernels, and it is a rare ear of sweet corn that is free from earworm, even with regular insecticide spray programs (Brewbaker and Mitchell Personal comunication).

The objectives of this study were to find high husk number germplasms and to determine gene action conditioning husk number. Preliminary studies were made to determine the significance of high husk numbers in corn earworm resistance.

MATERIALS AND METHODS

Preliminary observations for husk numbers were made using a high-lysine composite, 43 inbreds, and 37 maize races.

Heritability and combining ability estimates for husk numbers in corn were made with nine inbreds, comprising five tropical parents -- AA8, Ant2D, CM104, CM105, and CM111 -- and four temperate parents -- B37, CI21E, Mo17, and Oh545. A randomized complete block design with four replications was employed. Each replication consisted of a single row plot, each plot containing about 15 plants spaced 20 cm between plants and 75 cm between rows. Ten plants were used for the counting husk numbers at two weeks after mid-silking. Plots were treated with 600 lb/acre of 16-16-16 fertilizer before planting and 300 lb/acre of 16-4-4 fertilizer at about one month after emergence. All materials were grown at the Waimanalo Farm between June and September, 1973. The Waimanalo soil is a silty clay with pH 6.5. Griffing's methods 2 and 4, model I and II (Griffing 1956) were employed for estimates of heritability, and combining ability.

Results

1. Husk Number and Earworm Damage

Husk numbers and earworm damage were recorded at full maturity for 81 plants of high-lysine composite o_2COMP2 (Hawaii), harvested in February, 1974. All husks were counted, excluding the subtending bract. Earworm damages were estimated in two ways: (1) Number of kernels lost/total number of kernels per ear X 100 ("field corn method"), and (2) Damaged ear length/total ear length X 100 ("sweet corn method"). All

Table 59. Husk numbers, kernel damage, and earlength loss due to earworms in o_2COMP2 (Hawaii)

	Damage in percent			Damage in percent	
Husk Number	Kernel No.	Ear length	Husk Number	Kernel No.	Ear length
6	13.7	50.3	10	6.5	15.8
7	5.3	35.5	10	7.2	38.0
7	12.6	64.3	10	10.1	42.9
7	5.0	43.8	10	11.8	39.3
8	1.8	15.6	10	9.9	23.7
8	1.0	14.7	10	6.4	31.3
8	9.4	34.0	10	3.5	15.2
8	1.8	16.4	10	6.7	30.4
8	8.6	41.7	10	15.2	59.3
8	9.8	66.7	10	14.5	54.5
8	11.8	60.0	10	9.0	52.6
8	6.6	61.3	10	5.0	20.0
8	13.5	37.5	10	10.7	49.7
			10	12.6	52.9
9	2.3	29.7	10	9.1	44.4
9	6.3	27.1	10	10.1	61.9
9	6.7	20.0			
9	3.9	21.7	11	1.8	13.3
9	2.6	17.9	11	3.1	36.5
9	1.2	6.9	11	3.5	13.8
9	10.2	43.9	11	5.6	44.2
9	4.7	25.9	11	7.3	40.6
9	3.9	22.9	11	7.0	25.8
9	6.0	52.0	11	4.8	20.1
9	5.5	13.8	11	0.9	6.3
9	6.5	42.3	11	6.0	17.6
9	7.6	37.0	12	3.1	20.6
9	3.8	16.1	12	5.2	31.5
9	11.4	51.6	12	2.2	5.1
9	2.8	15.6	12	6.8	46.2
9	11.3	62.1	12	3.3	20.0
9	3.4	26.9	12	0.9	7.7
9	10.7	56.2	12	9.1	65.5
9	4.8	43.2			
9	7.9	42.3	13	2.0	12.2
9	11.4	56.4	13	1.2	6.7
9	10.1	41.4	13	0.4	1.4
10	0.4	8.9	13	2.5	11.4
10	2.6	21.2	13	0.3	2.6
10	4.2	17.9	13	5.9	50.0
10	8.0	45.5	14	4.5	25.0
10	8.3	40.6	14	2.7	39.5
			Avg. 9.9	6.3	32.7

percentage data were transformed into angles for calculations of correlations.

Husk numbers and earworm damages, presented as losses in kernel number and in ear length, are summarized in Table 59. Average husk number, earworm damage in (1) and (2) methods were 9.9, 6.3%, and 32.7%, respectively.

The correlation between husk number and kernel loss was $r = -.396^{**}$. The correlation between husk number and damaged ear length was $r = -.364^{**}$. Earworm resistance is considered particularly important for high-lysine corn, with soft floury kernels.

2. Husk Number, Leaf Number, and Days to mid-silk of Inbred Lines.

Forty three widely used US and foreign maize inbred lines were studied for husk numbers, leaf numbers, and days to mid-silk. Average husk numbers of the 43 inbreds are summarized in Table 60. Large variations were found in husk numbers. The average husk number of the 43 inbreds was 9.76 and ranged from 15.9 for CM111 to 6.8 for C123. Seven of the 43 lines had more than 12 husk numbers, while 12 lines had fewer than 8 husks. Most of the inbreds with high husk number originated from the tropics or southern temperate regions, and most of the lines with low husk number were from the temperate regions. Leaf number and days to mid-silk data were taken from the same materials and are also summarized in Table 60. In general, higher husk numbers were associated with later maturity ($r = .384^{*}$), reflecting the southerly or tropical nature of the lines with high husk number. High leaf number is also known to be associated with later maturity (Chase and Nanda 1965, 1969), and was significantly correlated here with higher husk numbers ($r = .549^{**}$).

Table 60. Average husk numbers, leaf numbers, and days to mid-silk of 43 inbred lines

Inbred	Husk number	Leaf number	Days to mid-silk
CM111	15.9	14.4	68
CI64	14.7	11.9	68
F6	14.5	15.3	67
CM105	14.4	13.6	64
Mo18W	13.7	12.0	65
CM104	12.7	13.3	65
F44	12.0	15.3	67
N6G	11.4	12.9	57
CI66	11.2	15.4	64
M14	11.2	11.6	58
R168	10.7	10.5	51
H95	10.5	11.2	56
Oh51A	10.4	10.7	52
A632	10.3	12.4	52
H84	10.3	13.1	61
A619	10.1	10.3	51
PH9	10.1	11.1	53
B14A	9.9	12.2	56
Va26	9.8	11.4	56
38-11	9.4	10.9	60
B68	9.4	12.9	60
Mo5	9.4	12.4	61
Oh545	9.4	10.7	54
AA8	9.2	11.2	58
Oh43	9.2	10.0	53
Ga209	9.0	13.6	63
N28	8.7	12.1	57
T232	8.7	11.7	63
Ky226W	8.4	11.8	65
Va35	8.4	11.1	59
H93	8.0	13.0	64
B37	7.8	11.8	60
H94	7.8	12.8	64
W64	7.7	9.0	56
B70	7.5	12.3	55
B73	7.5	12.2	56
CI21E	7.5	11.9	68
H60	7.5	9.4	58
Oh07A	7.4	11.8	61
H55	7.3	10.4	59
Mo17	7.0	11.6	58
C103D	6.8	10.0	59
C123	6.8	9.5	55
Average	9.76	11.92	59.5

While very few early inbreds had high husk number, several late inbred had low husk numbers, e.g. Ga209, T232, Ky226W, H93, and CI21E. It would appear that the association is causal, and puts no limitation on the breeder for reassociations, as e.g., producing early inbreds with higher husk number.

3. Husk Numbers of Maize Races

In one season, 37 maize races were tested and the average husk numbers from 10 individual plants of each are summarized in Table 61. Considerable variation was found in husk number, with average of 10.7 husks. The race Zapalote Grande, Cuban Flint, Haitian Yellow, Chococeno, and Costeno amarilla had more than 15 husks. Zapalote Grande had the highest husk number (19 husks). Races with fewer than 8 husks were largely highland popcorns, rarely subjected to severe earworm attack. The very primitive lowland popcorn race, Nal Tel, had 13 husks. A majority of the lowland races from Mexico and Caribbean (e.g. Zapalote, Cuban, Haitian, Tuxpeno, Vandeno) have high husk numbers associated with their recognized earworm resistance (Wellhausen et al. 1952, Robert et al. 1957, Grant et al. 1963).

4. Husk Numbers of 9 X 9 Diallel

Average husk numbers of 9 parents and their 36 F_1 hybrids are summarized in Table 62. The grand mean, parental and F_1 means were 11.02, 10.13, and 11.25 respectively. Large variations among parents and their F_1 hybrids were found in husk numbers (Table 62). The range of husk numbers of the nine parents was from 7.0 for Mo17 to 14.4 for CM111, and that of the 36 F_1 hybrids was from 8.4 for Oh545 X Mo17 to 15.8 for CM111 X CM104. Hybrid averages closely followed the parental

Table 61. Average husk numbers of 37 maize races.

Race	Husk number
Zapalote Grande	19.0
Cuban Flint 152	18.0
Haitian Yellow	16.3
Chococeno	16.1
Costeno amarilla	15.3
Cuareritino	14.7
Puya	14.4
Tuxpeno	13.4
Andaqui	13.0
Comiteco mezcla	13.0
Comun amarilla	13.0
Nal Tel	13.0
Kaurou	12.9
Negrito	12.9
Vandeno	12.7
Cariaco	11.5
Guirua	11.5
Comun blanco	11.0
Cacao amarillo	11.0
Chapalote	10.4
Avati Tupi	10.3
Chulpi	10.0
Conico norteno	10.0
Jucunas	10.0
Mal-Jo	9.7
Clavo	9.4
Bolita	9.0
Montana	9.0
Caingang	9.0
Cabuya amarilla	8.5
Calchaqui	8.2
Harinoso de Ocho	8.2
Amagaceno	7.9
Palomero Toluqueno	7.6
Imbricado	7.5
Lady Finger Pop	6.8
Sabanero blanco	6.0
Average	10.6

Table 62. Average husk number of 9 parents and their 36 F_1 hybrids

Parent	CM111	CM105	CM104	Ant2D	Oh545	AA8	CI21E	B37	Mo17	Avg. of hybrid
CM111	14.35	15.23	15.75	13.90	14.33	12.28	13.48	12.63	12.78	13.80
CM105		12.33	14.00	11.85	11.50	11.28	12.30	11.20	10.20	12.19
CM104			11.95	11.00	12.03	11.18	10.23	8.65	10.38	11.65
Ant2D				11.90	12.33	13.43	10.75	10.68	9.85	11.73
Oh545					9.43	10.08	9.90	9.95	8.35	11.06
AA8						8.78	10.23	8.80	8.83	10.76
CI21E							7.80	8.65	8.60	10.52
B37								7.58	8.45	9.88
Mo17									7.00	9.68

Grand mean = 11.02, Parental mean - 10.13, and F_1 mean = 11.25

se of treatment means based parent and F_1 data = 0.243

se of the difference between two treatment means = 0.344

means (r^2 = .933**, t = 9.84).

Four tropical parental lines, CM111, CM105, CM104, and Ant2D, had higher husk number than four temperate lines, Oh545, CI21E, B37, and Mo17. The Hawaiian sweet corn inbred AA8 had a low husk numbers. The highest husk number parent CM111 was bred by the Rockefeller Foundation, in India, from a yellow Cuban flint, Cuba 342. The lowest husk number parents Mo17 and B37 are widely used dent inbreds in the U. S. Corn Belt and many other regions.

Mean squares from the randomized block analysis of variance for parental and hybrid husk numbers are summarized in Table 63, based on Griffing's methods 2 and 4 and fixed model I. Highly significant differences were detected among 45 genotypes for husk numbers in both methods, a necessary condition for computation of the combining abilities summarized in Table 64.

General combining ability (GCA) and specific combining ability (SCA) mean squares were highly significant based on both methods 2 and 4 (Table 64). Variance component analyses and their respective percent values are summarized in Table 65. The percent values for GCA and SCA effects were 67.8 and 30.1 for method 2, and 73.6 and 24.2 for method 4, respectively. Environmental variances were extremely small, accounting for only 2.1% for method 2 and 2.2% for method 4. The results indicated that replication was adequate, and suggested little need for repetition of the experiment.

Application of the random model to these data was made on the assumption that the parents represent a truly random sample of maize inbreds in terms of husk numbers. On this model, GCA and SCA effects were also highly significant, based on error mean squares of 0.358

Table 63. Analyses of variance of husk number in Table 62, based on Griffing's methods 2 and 4, fixed model I

Source	Method 2 d.f.	Method 2 Mean Square	Method 4 d.f.	Method 4 Mean Square
Genotypes	44	172.439**	35	158.252**
Replication	3	43.909	3	43.751
Genotypes x rep.	132	14.317	105	5.643
Error	1620	2.364	1296	2.529

**p 0.01

Table 64. Combining ability mean squares based on methods 2 and 4, model I, for husk number

Source	Method 2 d.f.	Method 2 Mean Square	Method 4 d.f.	Method 4 Mean Square
GCA	8	21.020**	8	14.769**
SCA	36	0.906**	27	0.753**
Error	1620	0.059	1296	0.063

**p 0.01

Table 65. Variance components analysis for GCA and SCA values in Table 64

Component	Method 2 Variance	Method 2 % Value	Method 4 Variance	Method 4 % Value
GCA	1.906	67.78	2.101	73.63
SCA	0.847	30.12	0.689	24.16
Error	0.059	2.10	0.063	2.21

(Method 2) and 0.141 (Method 4), respectively.

Summarized in Table 66 are the phenotypic, genotypic, additive, non-additive, and environmental variances, as well as broad and narrow sense heritabilities. The genotypic variances were approximately 64.1% and 64.6% for methods 2 and 4, accounting for a major portion of the phenotypic variances of 6.57 and 7.15, respectively. Additive genetic variances were 86.9% and 86.8% for methods 2 and 4, respectively, and constituted a major portion of the total genotypic variance. Non-additive genetic variances constituted a very small portion of the total genotypic variance. Broad sense heritabilities were approximately 61% for both methods 2 and 4, and narrow sense heritabilities were approximately 52% for the two methods (Table 66). High narrow sense heritability estimates reflect the high additive genetic variance in the inheritance of husk numbers in maize.

General combining ability effects for the nine parents are summarized in Table 67, as calculated on the fixed model. As evident from the original data (Table 62), GCA effects for husk number differed widely among the parents. Inbreds CM111, CM105, CM104, and Ant2D had highly significant positive GCA effects, indicating that these four parents were good general combiners for increasing husk numbers in hybrid combinations. The remaining five parents had highly significant negative GCA effects. CM111 was the best combiner for husk number, increasing husk numbers by 2.6 husks (Method 2), on the average, in hybrid combinations with the remaining eight lines. In contrast, Mo17 decreased husk numbers by 1.7 (Method 2), on the average, in its hybrid combinations.

The coefficient of linear determination ($r^2 = 0.769$) between parental husk numbers and their respective GCA effects based on method 2

Table 66. Estimates of phenotypic variance and its components, and of broad (bH) and narrow (nH) sense heritabilities for husk number

Variance	Method 2	Method 4
Phenotypic	6.569 ± 1.923	7.145 ± 2.123
Genotypic	4.205 ± 1.924	4.616 ± 2.121
Additive	3.657 ± 1.911	4.005 ± 2.111
Non-additive	0.548 ± 0.218	0.611 ± 0.206
Environmental	2.364 ± 0.083	2.529 ± 0.099
bH	60.6 (49.1 - 72.1)	61.2 (49.7 - 72.7)
nH	51.6 (37.6 - 65.5)	51.9 (37.7 - 66.0)

Table 67. Estimates of general combining ability effects for husk number

Parent	Parental Mean	General Combining Ability Effects Method 2	Method 4
CM111	14.35	2.62	2.91
CM105	12.33	1.09	1.08
CM104	11.95	0.62	0.46
Ant2D	11.90	0.67	0.54
Oh545	9.43	-0.27	-0.22
AA8	8.78	-0.60	-0.56
CI21E	7.80	-0.96	-0.84
B37	7.58	-1.46	-1.57
Mo17	7.00	-1.71	-1.80
se_d		0.104	0.134

was highly significant.

Specific combining ability effects of the 36 F_1 hybrids based on methods 2 and 4 are summarized in Table 68. Some differences occurred among individual SCA effects calculated by the two different methods. Hybrid AA8 X Ant2D showed the highest SCA effects for increasing husk numbers, having 2.34 (Method 2) or 2.20 (Method 4) more husks than expected on the basis of the GCA effects. In contrast, hybrid CM104 X B37 showed the lowest SCA effect with 1.54 (Method 2) or 1.49 (Method 4) fewer husks than expected.

Heterotic effects for husk number (HN) were calculated from original data (Table 62) by

$$(F_1 HN - \text{Mid-parent HN}) \;/\; \text{Mid-parent HN} \times 100$$

and are summarized in Table 68. The average heterotic effect was 11.18% (Table 69). Most of the F_1 hybrids showed positive heterotic effects except Ant2D X CM104, Ant2D X CM105, and B37 X CM104. Wide variations in heterotic effects were found among 36 F_1 hybrids. Hybrid CI21E X CM104 showed the highest heterotic effects, while hybrid B37 X CM104 showed the lowest heterotic effects.

Table 68. Estimates of specific combining ability effects for husk number (Method 2 results above diagonal; Method 4 below)

Parent	CM111	CM105	CM104	Ant2D	Oh545	AA8	CI21E	B37	M017
CM111		0.50	1.49	-0.41	0.95	-0.77	0.79	0.45	0.84
CM105	-0.01		1.27	-0.93	-0.34	-0.24	1.15	0.55	-0.20
CM104	1.13	1.21		-1.32	0.65	0.13	-0.47	-1.54	0.44
Ant2D	-0.80	-1.02	-1.25		0.90	2.34	0.02	0.45	-0.13
Oh545	0.39	-0.61	0.54	0.76		-0.08	0.10	0.66	-0.70
AA8	-1.32	-0.49	0.03	2.20	-0.39		0.76	-0.16	0.11
CI21E	0.16	0.81	-0.64	-0.20	-0.29	0.37		0.05	0.24
B37	0.04	0.44	-1.49	0.46	0.49	-0.32	-0.19		0.60
Mo17	0.41	-0.33	0.46	-0.14	-0.88	-0.07	-0.02	0.57	

se_d between effects of two parent lines a based on method 2 = 0.0274

se_d between effects of two crosses having one parent line in common:

Method 2 = 0.328
Method 4 = 0.329

se_d between effects of two crosses having no parent lines in common:

Method 2 = 0.311
Method 4 = 0.301

Table 69. Estimates of heterotic effects in percent for husk number

Parent	CM105	CM104	Ant2D	Oh545	AA8	CI21E	B37	Mo17
CM111	14.17	19.77	5.90	20.52	6.18	21.72	15.18	19.72
CM105		15.32	-2.19	5.69	6.87	22.21	12.51	5.54
CM104			-7.76	12.54	7.86	35.95	11.42	9.55
Ant2D				15.61	29.88	9.14	9.65	4.23
Oh545					10.70	14.92	16.99	1.64
AA8						23.40	7.58	11.91
CI21E							12.48	16.22
B37								15.91

Average heterotic effect in percent = 11.18

DISCUSSION

Highly significant correlation values of husk number and earworm damage, considering both kernel damage and ear length reduction, indicated that high husk numbers can reduce earworm damage substantially. Collins and Kempton (1917) reported no significant correlation of husk number and earworm damage ($r = -.08$), a report based on Corn Belt stocks with much less genetic variability for husk number than these studied here. This early results may have led to the view that husk number is not an important factor to reduce earworm damage, as little subsequent research on husk number has been reported.

Parental inbred lines used in these study were extremely diverse temperate and tropical maize lines. All parental lines with high husk number were from tropical maize germplasms. Poole (1945) reported that the highest resistance to earworm was associated with southern dent germplasm (of Tuxpeno origin, a high husk number race). Most of the parental lines with low husk numbers were from temperate regions. Parent Mo17 of the diallel has been reported as highly susceptible to earworms (Straub et al. 1973). Dicke and Jenkins (1945) observed that flinty endosperm with hard texture near the tip of the ear offered the best protection against earworm damage.

Two high husk number parents, CM111 and CM105, showed exceptionally tight and thick husks, and appear to be promising sources of earworm resistance. The importance of the tight and thick husks to prevent earworm damage has been reported (Collins and Kempton 1917, Phillips and Barker 1931. Burk et al. 1936, Dicke and Jenkins 1945, Del Valle and Miller 1963, Painter 1968, and Widstrom et al. 1970). Widstrom et al.

(1970) obtained a significant correlation (r = -.33) between husk tightness and earworm damage.

Maize race Zapalote Grande had the highest husk number, 19. Straub et al. (1973) reported that Zapalote Chico was highly resistant to earworms, and suggested that the Zapalotes contained some antibiotic material in kernel or husks. Husk numbers in maize races have been reported by Wellhausen et al. (1952) in Mexican races, by Robert et al. (1957) in Colombia races, and by Grant et al. (1963) in Venezuela races. Wellhausen et al. reported the highest husk numbers for Zapalote Grande (18.2) and the lowest for Chalqueno (7.2). The following maize races had husk numbers as follows: Tuxpeno (15.6), Vandena (12.4), Nal Tel (11.4), Comiteco mezcla (10.6), Bolita (8.6), and Harinoso de Ocho (8.8), also reported in the present study (Table 60). Robert et al. found the highest husk number in Andaqui (16.6) and the lowest in Imbricado (8.2), respectively (cf. Table 60). Grant et al. found the highest husk number in Chirimito, accounting for 20.2, and the lowest in Pollo (8.2), with 18.3 husks in Cariaco.

General combining ability mean squares for husk number were substantially larger than specific combining ability mean squares, and narrow sense heritability was 52%. High general combining ability effects for earworm resistance have been reported (Widstrom and Hamm 1969). Widstrom et al. (1972) estimated high additive genetic variances for earworm resistance in three composite populations derived from Latin American sources over dominance variances. They observed high heritability and McMillian (1973) found that additive, dominance, and epistatic variances were all significant for earworm resistance in crosses between field and sweet corns.

Most of the parents with high husk numbers in this study belong to the late maturity group. However, in the diallel study, inbred CI21E had only 7.8 husks, but it was one of the latest to mature in Hawaii. Collins and Kempton (1917) and Widstrom and Starks (1966) reported a significant correlation between earworm damage and maturity. However, Yarnell (1952) observed no apparent effect of maturity on earworm susceptibility.

SUMMARY

Studies were made to assess the effect of rust on maize yield in Hawaii, to assess the resistance of widely used inbreds and races, to determine gene action conditioning generalized resistance, to survey host-pathogen interactions, and to determine gene action conditioning husk number in maize.

P. sorghi affected nine agronomic characteristics significantly at the 1% level. Average per cent reductions due to rust over two seasons were 31% (grain yield), 27% (total fresh plant weight with ear), 10% (1000 kernel weight), 11% (ear length), 10% (ear diameter), 3% (plant height), 5% (ear height), and 1.4 days (mid-silking). The significant yield reductions obtained in this study give an indication of the potential damage that may be caused by P. sorghi in maize in subtropical as well as temperate regions.

P. sorghi races present in the maize nurseries at the Waimanalo Farm were shown to be pathogenic on all international monogenic sources except only six Rp_1 sources a, d, d(2), f, g, and k.

Most widely used US maize inbreds were rated as highly susceptible to P. sorghi. Two of forty inbreds showed outstanding generalized resistance. They were Oh545 and CM105. Highly generalized resistant inbreds showed a tendency toward resistance at one week before mid-silking and continued to exhibit this ontogenetic change until two weeks following mid-silking. Forty-one maize races exhibited differing degrees of resistance. Sweet corn inbred 677a resistance was interpreted as monogenic, recessive, and an allele of one of the five Rp loci located on chromosome 10 in maize.

Gene action conditioning generalized resistance was determined by diallel and generation mean analyses. Highly significant general (GCA) and specific (SCA) combining ability mean squares were obtained for rust resistance in the diallel crosses, with GCA always substantially larger than SCA. Broad and narrow sense heritabilities were 85% and 72%, respectively. Results from the generation mean analyses confirmed the major contribution of GCA effects expressed as the importance of additive genetic variance for rust resistance. However, high non-additive genetic variance was observed primarily in crosses involving moderately resistant sources. The calculated minimum numbers of gene pairs controlling generalized resistance revealed that relatively small numbers of gene pairs, approximately two, are present. It is recommended that a breeding program for selection of lines or crosses that contribute polygenic rust resistance based on highly additive gene action would be desirable to keep P. sorghi a pathogen of minor economic importance in maize.

Heritabilities for lesion length due to P. sorghi were high accounting for 63% for broad sense and 57% for narrow sense, respectively. Additive genetic variance was a major portion of the total genotypic variance, accounting for 93%. The heterotic effect was extremely high, averaging 37%. It was evidenced that lesion length was inherited independently from pustule number. Parental lines having long lesions should be excluded from selection for rust resistance in breeding programs. Generalized resistance was accompanied by fewer pustules at early vegetative stages.

Peroxidase activity was increased significantly by P. sorghi infection after 8 days, but not after 2 days. The increased peroxidase activity was not directly related with generalized resistance to rust. Rust

resistant maize plants controlling monogenic dominant Rp_1^d gene showed stronger peroxidase responses than their susceptible counterparts.

Husk number in a maize composite was significantly correlated with earworm damage ($r = -.4^{**}$). Husk numbers in the 43 inbred lines were also significantly correlated with leaf number ($r = .6^{**}$) and days to mid-silk ($r = .4^{*}$). Five of 37 maize races averaged more than 15 husks and two races had fewer than 7 husks. Zapalote Grande had the highest husk number, 19. Inbreds CM111 and CM105 had high husk numbers with high GCA effects. Broad and narrow sense heritability estimates were 61% and 51%, respectively. It should be possible to increase husk number and improve earworm resistance by various breeding methods, capitalizing on additive genetic variance.

APPENDIX

Table 1. Average, variance, standard deviation, associated standard error of each generation and observed frequency distributions of corn plants based on the scale 1 to 7 for rust ratings

Gen.	$\bar{x}$	V	S	s_e	Rust ratings 1	2	3	4	5	6	7
					Oh545 (P_r) x AA8 (P_s)						
P_r	1.36	0.233	0.483	0.058	45	25	-	-	-	-	-
P_s	6.80	0.162	0.402	0.048	-	-	-	-	-	14	56
F_1	3.31	0.306	0.553	0.066	-	3	42	25	-	-	-
F_2	3.73	2.426	1.558	0.093	8	70	64	40	52	37	9
B_r	2.70	1.350	1.162	0.080	33	63	68	26	20	-	-
B_s	3.90	1.392	1.180	0.081	2	25	49	71	42	21	-
					Oh545 (P_r) x B37 (P_s)						
P_r	1.36	0.233	0.483	0.058	45	25	-	-	-	-	-
P_s	6.90	0.091	0.302	0.036	-	-	-	-	-	7	63
F_1	3.03	0.260	0.510	0.061	-	7	55	7	1	-	-
F_2	3.78	2.193	1.481	0.088	11	45	86	39	57	35	7
B_r	2.38	1.011	1.005	0.069	38	89	58	16	9	-	-
B_s	4.17	1.235	1.111	0.076	-	15	46	62	65	24	-
					Oh545 (P_r) x C123 (P_s)						
P_r	1.36	0.233	0.483	0.058	45	25	-	-	-	-	-
P_s	6.11	0.364	0.603	0.072	-	-	-	-	9	44	17
F_1	2.86	0.211	0.460	0.055	-	13	54	3	-	-	-
F_2	3.82	1.918	1.385	0.083	10	46	68	47	78	31	-
B_r	2.38	0.914	0.956	0.066	32	95	57	17	7	-	-
B_s	4.24	1.716	1.309	0.094	1	19	44	40	49	41	-
					Oh545 (P_r) x CM104 (P_s)						
P_r	1.36	0.233	0.483	0.058	45	25	-	-	-	-	-
P_s	6.73	0.201	0.448	0.054	-	-	-	-	-	19	51
F_1	3.30	0.329	0.574	0.069	-	4	41	25	-	-	-
F_2	3.36	1.973	1.405	0.084	9	82	87	33	44	20	5
B_r	2.69	0.995	0.998	0.070	15	83	67	25	13	-	-
B_s	3.77	1.329	1.153	0.076	1	42	38	90	45	14	-

Table 1. (Continued) Average, variance, standard deviation, associated standard error of each generation and observed frequency distributions of corn plants based on the scale 1 to 7 for rust ratings

Gen.	$\bar{x}$	V	S	s_e	Rust ratings 1	2	3	4	5	6	7
					Oh545 (P_r) x H95 (P_s)						
P_r	1.36	0.233	0.483	0.058	45	25	-	-	-	-	-
P_s	4.79	0.200	0.447	0.053	-	-	-	-	53	1	-
F_1	2.37	0.237	0.487	0.058	-	44	26	-	-	-	-
F_2	2.78	1.420	1.192	0.071	33	94	92	30	25	6	-
B_r	2.26	0.919	0.959	0.067	40	107	37	21	5	-	-
B_s	3.48	1.390	1.179	0.083	4	49	49	47	54	-	-
					CM105 (P_r) x AA8 (P_s)						
P_r	1.73	0.259	0.509	0.061	21	47	2	-	-	-	-
P_s	6.80	0.162	0.403	0.048	-	-	-	-	-	14	56
F_1	4.25	0.496	0.704	0.091	-	-	9	27	24	-	-
F_2	4.50	2.240	1.497	0.076	2	32	93	48	99	76	35
B_r	3.50	1.828	1.352	0.094	9	42	69	31	44	11	3
B_s	5.30	1.707	1.307	0.109	-	6	11	12	46	43	26
					CM105 (P_r) x B37 (P_s)						
P_r	1.73	0.259	0.509	0.061	21	47	2	-	-	-	-
P_s	6.90	0.091	0.302	0.036	-	-	-	-	-	7	63
F_1	3.49	0.369	0.608	0.073	-	-	40	26	4	-	-
F_2	4.39	2.512	1.585	0.088	3	37	72	56	68	48	39
B_r	3.46	1.646	1.283	0.089	4	50	69	35	39	11	2
B_s	5.00	1.907	1.381	0.094	-	7	32	33	62	48	35
					CM105 (P_r) x C123 (P_s)						
P_r	1.73	0.259	0.509	0.061	21	47	2	-	-	-	-
P_s	6.11	0.364	0.603	0.072	-	-	-	-	9	14	17
F_1	3.07	0.421	0.649	0.078	-	12	40	17	-	-	-
F_2	3.97	1.989	1.410	0.084	9	32	76	51	67	39	5
B_r	3.29	1.621	1.273	0.088	14	49	63	35	45	4	-
B_s	3.72	1.647	1.284	0.087	7	40	40	65	51	15	-

Table 1. (Continued) Average, variance, standard deviation, associated standard error of each generation and observed frequency distributions of corn plants based on the scale 1 to 7 for rust ratings

Gen.	$\bar{x}$	V	S	s_e	Rust Ratings 1	2	3	4	5	6	7
					CM105 (P_r) x CM104 (P_s)						
P_r	1.73	0.259	0.509	0.061	21	47	2	-	-	-	-
P_s	6.73	0.201	0.448	0.054	-	-	-	-	-	19	51
F_1	4.27	0.288	0.536	0.064	-	-	2	48	19	1	-
F_2	4.32	2.302	1.517	0.078	4	41	91	64	89	59	33
B_r	3.75	1.967	1.403	0.097	7	43	45	37	56	22	-
B_s	5.13	1.719	1.311	0.097	-	-	21	45	44	35	38
					CM105 (P_r) x H95 (P_s)						
P_r	1.73	0.259	0.509	0.061	21	47	2	-	-	-	-
P_s	4.79	0.200	0.447	0.053	-	-	-	16	53	1	-
F_1	4.27	0.436	0.660	0.085	-	-	7	30	23	-	-
F_2	3.87	2.150	1.466	0.081	7	55	93	51	69	40	11
B_r	3.26	1.697	1.303	0.091	9	62	56	34	35	10	-
B_s	4.05	1.493	1.222	0.084	1	20	57	54	51	30	-
					CM111 (P_r) x AA8 (P_s)						
P_r	2.71	0.294	0.542	0.065	-	23	44	3	-	-	-
P_s	6.80	0.162	0.402	0.048	-	-	-	-	-	14	56
F_1	5.11	0.364	0.603	0.072	-	-	-	9	44	17	-
F_2	4.73	2.217	1.489	0.083	1	20	69	34	87	71	39
B_r	4.34	1.905	1.380	0.096	-	23	41	40	58	36	9
B_s	5.39	1.693	1.301	0.090	-	6	17	25	44	78	40
					CM111 (P_r) x B37 (P_s)						
P_r	2.71	0.294	0.542	0.065	-	23	44	3	-	-	-
P_s	6.90	0.091	0.302	0.036	-	-	-	-	-	7	63
F_1	4.40	0.301	0.549	0.066	-	-	2	38	30	-	-
F_2	4.67	2.350	1.495	0.084	1	24	60	50	80	63	39
B_r	4.52	2.279	1.510	0.104	1	18	48	31	46	47	19
B	5.72	1.464	1.210	0.086	-	-	17	13	39	69	61

Table 1. (Continued) Average, variance, standard deviation, associated standard error of each generation and observed frequency distributions of corn plants based on the scale 1 to 7 for rust ratings

Gen.	$\bar{x}$	V	S	s_e	Rust ratings 1	2	3	4	5	6	7
					CM111 (P_r) x C123 (P_s)						
P_r	2.71	0.294	0.542	0.065	-	23	44	3	-	-	-
P_s	6.11	0.364	0.603	0.072	-	-	-	-	9	44	17
F_1	4.50	0.399	0.631	0.075	-	-	4	28	37	1	-
F_2	4.37	2.285	1.512	0.092	5	22	63	52	59	45	24
B_r	4.28	2.410	1.554	0.107	1	34	44	23	55	40	13
B_s	4.96	1.251	1.119	0.082	-	-	31	24	56	73	13
					CM111 (P_r) x CM104 (P_s)						
P_r	2.71	0.294	0.542	0.065	-	23	44	3	-	-	-
P_s	6.73	0.201	0.448	0.054	-	-	-	-	-	19	51
F_1	4.81	0.269	0.519	0.062	-	-	-	17	49	4	-
F_2	4.21	2.567	1.602	0.089	5	49	72	58	55	58	27
B_r	3.99	1.918	1.385	0.096	-	35	51	44	50	22	8
B_s	5.75	1.546	1.243	0.087	-	-	16	16	47	52	75
					CM111 (P_r) x H95 (P_s)						
P_r	2.71	0.294	0.542	0.065	-	23	44	3	-	-	-
P_s	4.79	0.200	0.447	0.053	-	-	-	16	53	1	-
F_1	3.39	0.240	0.490	0.059	-	-	43	27	-	-	-
F_2	4.39	2.175	1.475	0.082	2	28	74	71	58	62	26
B_r	4.00	1.718	1.311	0.090	-	27	62	39	53	25	4
B_s	4.59	1.191	1.091	0.075	-	8	26	50	67	49	-
					Mo17 (P_r) x AA8 (P_s)						
P_r	3.37	0.352	0.594	0.071	-	-	48	18	4	-	-
P_s	6.80	0.162	0.402	0.048	-	-	-	-	-	14	56
F_1	4.53	0.311	0.557	0.067	-	-	2	29	39	-	-
F_2	4.95	1.629	1.276	0.076	-	8	38	45	91	71	30
B_r	4.59	1.497	1.224	0.084	-	6	39	52	65	35	13
B_s	5.45	1.364	1.168	0.079	-	-	14	37	46	79	42

Table 1. (Continued) Average, variance, standard deviation, associated standard error of each generation and observed frequency distributions of corn plants based on the scale 1 to 7 for rust ratings

Gen.	$\bar{x}$	V	S	s_e	Rust ratings 1	2	3	4	5	6	7
					Mo17 (P_r) x B37 (P_s)						
P_r	3.37	0.352	0.594	0.071	-	-	48	18	4	-	-
P_s	6.90	0.091	0.302	0.036	-	-	-	-	-	7	63
F_1	6.19	0.211	0.460	0.055	-	-	-	-	2	53	15
F_2	4.76	1.683	1.297	0.077	-	9	49	56	82	64	24
B_r	4.58	1.719	1.311	0.090	-	8	42	50	60	31	19
B_s	5.84	1.070	1.034	0.072	-	-	8	13	44	84	60
					Mo17 (P_r) x C123 (P_s)						
P_r	3.37	0.352	0.594	0.071	-	-	48	18	4	-	-
P_s	6.11	0.364	0.603	0.072	-	-	-	-	9	44	17
F_1	4.87	0.172	0.414	0.050	-	-	-	11	57	2	-
F_2	4.70	1.160	1.077	0.067	-	3	45	42	108	56	4
B_r	4.44	1.651	1.285	0.088	-	17	37	47	63	42	6
B_s	5.44	1.190	1.090	0.079	-	-	15	19	48	83	25
					Mo17 (P_r) x CM104 (P_s)						
P_r	3.37	0.352	0.594	0.071	-	-	48	18	4	-	-
P_s	6.73	0.201	0.448	0.054	-	-	-	-	-	19	51
F_1	4.86	0.240	0.490	0.059	-	-	-	14	52	4	-
F_2	5.14	1.706	1.306	0.073	-	6	37	49	82	98	46
B_r	4.46	1.713	1.309	0.119	-	5	22	43	26	11	13
B_s	5.62	1.152	1.073	0.075	-	-	8	23	53	74	46
					Mo17 (P_r) x H95 (P_s)						
P_r	3.37	0.352	0.594	0.071	-	-	48	18	4	-	-
P_s	4.79	0.200	0.447	0.053	-	-	-	16	53	1	-
F_1	4.09	0.369	0.608	0.073	-	-	10	44	16	-	-
F_2	4.31	1.263	1.124	0.067	-	6	71	83	75	39	6
B_r	3.87	1.382	1.176	0.082	-	23	69	45	51	19	-
B_s	4.88	1.042	1.021	0.076	-	-	22	40	57	63	-

Table 1. (Continued) Average, variance, standard deviation, associated standard error of each generation and observed frequency distributions of corn plants based on the scale 1 to 7 for rust ratings

Gen.	$\bar{x}$	V	S	s_e	Rust ratings 1	2	3	4	5	6	7
					CI21E (P_r) x AA8 (P_s)						
P_r	4.04	0.389	0.624	0.075	-	-	12	43	15	-	-
P_s	6.80	0.162	0.402	0.048	-	-	-	-	-	14	56
F_1	6.13	0.288	0.536	0.064	-	-	-	-	6	49	15
F_2	6.16	0.843	0.919	0.052	-	-	5	13	43	121	136
B_r	5.86	0.951	0.975	0.064	-	-	4	18	48	94	65
B_s	6.24	0.703	0.838	0.060	-	-	-	12	15	84	86
					CI21E (P_r) x B37 (P_s)						
P_r	4.04	0.389	0.624	0.075	-	-	12	43	15	-	-
P_s	6.90	0.091	0.302	0.036	-	-	-	-	-	7	63
F_1	6.17	0.434	0.659	0.079	-	-	-	-	10	38	22
F_2	6.01	0.954	0.977	0.054	-	-	2	27	62	113	124
B_r	5.71	1.056	1.027	0.071	-	-	5	21	55	75	52
B_s	6.19	0.738	0.859	0.059	-	-	-	13	22	87	88
					CI21E (P_r) x C123 (P_s)						
P_r	4.04	0.389	0.624	0.075	-	-	12	43	15	-	-
P_s	6.11	0.364	0.603	0.072	-	-	-	-	9	44	17
F_1	5.13	0.259	0.509	0.061	-	-	-	5	51	14	-
F_2	5.63	1.123	1.060	0.063	-	-	15	19	81	105	60
B_r	5.17	1.232	1.110	0.077	-	-	16	44	58	69	21
B_s	5.75	0.872	0.934	0.065	-	-	6	13	47	102	40
					CI21E (P_r) x CM104 (P_s)						
P_r	4.04	0.389	0.624	0.075	-	-	12	43	15	-	-
P_s	6.73	0.201	0.448	0.054	-	-	-	-	-	19	51
F_1	5.33	0.253	0.503	0.060	-	-	-	1	45	24	-
F_2	5.69	1.143	1.069	0.061	-	2	9	31	72	122	73
B_r	5.71	1.097	1.047	0.073	-	-	4	21	66	58	59
B_s	5.74	1.075	1.037	0.072	-	-	4	24	51	75	56

Table 1. (Continued) Average, variance, standard deviation, associated standard error of each generation and observed frequency distributions of corn plants based on the scale 1 to 7 for rust ratings

Gen.	$\bar{x}$	V	S	s_e	Rust ratings 1	2	3	4	5	6	7
					CI21E (P_r) x H95 (P_s)						
P_r	4.04	0.389	0.624	0.075	-	-	12	43	15	-	-
P_s	4.79	0.200	0.447	0.053	-	-	-	16	53	1	-
F_1	4.73	0.230	0.479	0.057	-	-	-	20	49	1	-
F_2	4.96	1.709	1.307	0.074	-	12	46	37	98	94	29
B_r	5.05	1.160	1.077	0.074	-	-	17	50	63	65	15
B_s	4.85	0.943	0.971	0.067	-	-	22	49	80	57	2

LITERATURE CITED

Andreev, L. N. and M. Shaw. 1969. A note on the effect of rust infection on peroxidase isozyme in flax. Canad. Jour. Bot. 43:1479-1483.

Anonymous. 1965. Losses in agriculture. U. S. Dept. Agr. ARS. Handbook No. 291. p. 42.

Arndt, C. H. and M. B. Stevenson. 1946. Unusual prevalence of corn rust in South Carolina. Plant Dis. Reptr. 30:427.

Arthur, J. C. 1934. Manual of the rusts in United States and Canada XV:116-117. Purdue Research Foundation, Lafayette, Indiana.

Biffen, R. H. 1905. Mendel's laws of inheritance and wheat breeding. Jour. Agr. Sci. 1:4-48.

Blanchard, R. A., J. H. Bigger, and R. O. Snelling. 1941. Resistance of corn strains to the corn earworm. Agron. Jour. 33:344-350.

Blanchard, R. A. and A. F. Satterthwaite. 1942. Manual infestation of corn strains as a method of determining differential earworm damage. Jour. Econ. Entomol. 35:508-511.

Borlaug, N. E. 1964. Puccinia sorghi on corn in Mexico (abstract). Phytopathology 36:395.

_____. 1965. Wheat, rust, and people. Phytopathology 55:1088-1098.

Brewbaker, J. L. and Y. Hasegawa. 1974. Polymorphisms of the 12 major peroxidases of maize. Proceedings, 3rd International Conference on Isozymes. Yale Univ., New Haven, Connecticut, Academic Press, N. Y. (In Press).

Brewbaker, J. L., S. K. Kim, and F. Laemmlen. 1972. Resistance to corn rust in Hawaii. Hawaii Agronomy, University of Hawaii.

Brewbaker, J. L., M. D. Upadhya, Y. Makinen, and T. MacDonald. 1968. Isoenzyme polymorphism in flowering plants. III. Gel electrophoretic methods and applications. Physiol. Plantarum 21:930-940.

Burk, E. F., Cross, C. B., and E. Hixson. 1936. Variety tests of sweet corn and its resistance to corn earworm and smut injury. Proc. Amer. Soc. Hort. Sci. 3:502-504.

Caldwell, R. M. 1966. Advances and challenges in the control of plant diseases through breeding. pp. 117-126. In: Pest Control by

Chemical, Biological, Genetic, and Physical Means. (Proc., AAAS Symposium). U. S. Dept. Agr., ARS. Misc. Publ. 33-110.

Call, L. E. 1932. The corn earworm and other insects injurious to corn. Kans. Agr. Expt. Sta. 6th Bien. Rep. Div. 1930-32:90-91.

Cammack, R. H. 1954. Observations on Puccinia polysora Underw. in West Africa. West African Maize Rust Research Unit Annual Rept. 1 (1953):16-31.

Castle, W. E. 1921. A method of estimating the number of genetic factors in cases of blending inheritance. Science 54:93-95.

Castle, W. E. and S. Wright. 1921. An improved method of estimating the number of genetic factors concerned in cases of blending inheritance. Science 54:223.

Chase, S. S. and D. K. Nanda. 1965. Relation between leaf number and maturity in maize (Zea mays). Amer. Jour. Bot. 52:628.

_____. 1967. Number of leaves and maturity classification in Zea mays L. Crop. Sci. 7:431-432.

Collins, G. N. and J. H. Kempton. 1917. Breeding sweet corn resistant to the corn earworm. Jour. Agr. Res. 11:549-572.

Comstock, R. E. and H. F. Robinson. 1952. Estimates of average dominance of genes. pp. 494-516. Heterosis. Iowa State Coll. Press, Ames, Iowa.

Coons, G. H. 1953. Breeding for resistance to disease. In Plant Diseases, The Yearbook of Agriculture, U. S. Dept. of Agric., 174-192.

Cummins, G. B. 1941. Identity and distribution of three rusts of corn. Phytopathology 31:856-857.

Daly, J. M. 1972. The use of near-isogenic lines in biochemical studies of the resistance of wheat to stem rust. Phytopathology 62:392-400.

Day, P. R. 1974. Genetics of Host-Parasite Interaction. W. H. Freeman and Company, San Francisco. 238 pp.

Del Valle, C. G. and J. C. Miller. 1963. Influence of husk length and tightness against corn earworm damage in sweet corn hybrids. Proc. Amer. Soc. Hort. Sci. 83:531-535.

Dicke, F. F. and M. T. Jenkins. 1945. Susceptibility of certain strains of field corn in hybrid combinations to damage by corn earworm. U. S. Dept. Agr. Tech. Bul. 898:36.

Dickson, J. G. 1952. General corn rust and leaf blight infection in Wisconsin. Plant Dis. Reptr. 36:350.

_____. 1956. Diseases of Field Crops. McGraw-Hill Company, N. Y.

Dickson, J. G., P. M. LeRoux, R. Syamananda, and A. L. Flangas. 1956. Progress reports on genetics of pathogenicity of Puccinia sorghi (Abstract). Phytopathology 46:11.

Douglas, W. A. 1947. The effect of husk extension and tightness on earworm damage to corn. Jour. Econ. Entomol. 40:661-664.

Dudley, J. W. and R. H. Moll. 1969. Interpretation and use of estimates of heritability and genetic variances in plant breeding. Crop Sci. 9:257-261.

Dunlap, A. A. 1937. Rust on teosinte and wilt of pepper in Connecticut. Plant Dis. Reptr. 21:426.

Eberhart, S. A. and A. R. Hallauer. 1968. Genetic effects for yield in single, three-way, and double-cross maize hybrids. Crop Sci. 8:377-379.

Eberhart, S. A. and W. A. Russell. 1969. Yield and stability for a 10-line diallel of single-cross and double-cross maize hybrids. Crop Sci. 9:357-361.

Farkas, G. L. and F. Solymosy. 1965. Host metabolism and symptom production in virus-infected plants. Phytopathology 54:85-93.

Farkas, G. L. and M. A. Stahmann. 1966. On the nature of changes in peroxidase isozymes in bean leaves infected by southern bean mosaic virus. Phytopathology 56:669-677.

Fehrmann, H. and A. E. Dimond. 1967. Peroxidase activity and Phytophthora resistance in different organs of the potato plant. Phytopathology 57:69-72.

Flangas, A. L. and J. G. Dickson. 1961. The genetic control of pathogenicity, serotypes and variability in Puccinia sorghi. Amer. Jour. Bot. 48:275-285.

Flor, H. H. 1955. Host-parasite interaction in flax rust: Its genetics and other implications. Phytopathology 45:680-685.

Frank, J. A. and J. D. Paxton. 1970. Time sequence of phytoalexin production in Harasoy and Harosoy 63 soybeans. Phytopathology 60:315-318.

Gamble, E. E. 1962. Gene effects in corn (Zea mays L.). I. Separa-

tion and relative importance of gene effects for yield. Canad. Jour. Plant Sci. 42:339-348.

Grant, U. J., W. H. Hatheway, D. H. Timothy, C. Cassalett D., and L. M. Roberts. 1963. Races of Maize in Venezuela. National Academy of Science-National Research Council. Pub. 1136, Wash. D. C.

Griffing, B. 1956. Concept of general and specific combining ability in relation to diallel crossing systems. Australian Jour. Biol. Sci. 9:463-493.

Gritton, E. T., G. L. Jones, N. T. Powell, and D. F. Matzinger. 1965. Inheritance of resistance to Fusarium wilt in flue-cured tobacco. Crop Sci. 5:547-550.

Grogan, C. O. and E. E. Rosenkranz. 1968. Genetics of host reaction to corn stunt virus. Crop Sci. 8:251-254.

Hagan, W. L. and A. L. Hooker. 1965. Genetics of reaction to Puccinia sorghi in eleven corn inbred lines from Central and South America. Phytopathology 55:193-197.

Hare, R. C. 1966. Physiology of resistance to fungal diseases in plants. Bot. Rev. 32:95-136.

Hawthorn, L. R. and R. K. Fletcher. 1934. Earworm damage in sweet corn varieties. Proc. Amer. Soc. Hort. Sci. 30:550-553.

Hilu, H. M. 1965. Host-pathogen relationships of Puccinia sorghi in nearly isogenic resistant and susceptible seedling corn. Phytopathology 55:563-569.

Holbert, J. R., W. L. Burlison, B. Koehler, C. M. Woodworth, and G. H. Dungan. 1924. Corn root, stalk, and ear rot diseases and their control through seed selection and breeding. Ill. Agr. Exp. Sta. Bul. 255.

Hooker, A. L. 1955. Corn diseases in Iowa nurseries in 1953 and 1954. Plant Dis. Rptr. 39:381-383.

_____. 1962a. Additional sources of resistance to Puccinia sorghi in the United States. Plant Dis. Rptr. 46:14-16.

_____. 1962b. Corn leaf disease. Proc., 17th Ann. Hybrid Corn Industry-Research Conf. 17:24-26.

_____. 1963. A second major gene locus in corn conditioning resistance to Puccinia sorghi. Phytopathology 53:221-223.

_____. 1967a. Disease and yield barriers. Proc., 22th Ann.

Hybrid Corn Industry-Research Conf. 17:24-36.

Hooker, A. L. 1967b. The genetics and expression of resistance in plants to rusts of the genus Puccinia. Ann. Rev. Phytopathology 5:163-182.

_____. 1967c. Inheritance of mature plant resistance to rust in corn (Abstract). Phytopathology 57:815.

_____. 1969. Widely based resistance to rust in corn. Iowa Agr. and Home Econ. Exp. Sta. Special Report 64:28-43.

_____. 1973. Maize. In: Breeding Plants for Disease Resistance, R. R. Nelson, ed. The Penn. State Univ. Press. pp. 132-154.

Hooker, A. L. and P. M. LeRoux. 1957. Sources of protoplasmic resistance to Puccinia sorghi in corn. Phytopathology 47:187-191.

Hooker, A. L. and W. A. Russell. 1962. Inheritance of resistance to Puccinia sorghi in six corn inbred lines. Phytopathology 52:122-128.

Hooker, A. L. and K. M. Saxena. 1967. Apparent reversal of dominance of a gene in corn for resistance to Puccinia sorghi. Phytopathology 57:1372-1374.

_____. 1971. Genetics of disease resistance in plants. Ann. Rev. of Genetics 5:407-424.

Hooker, A. L., G. F. Sprague, and W. A. Russell. 1955. Resistance to rust (Puccinia sorghi) in corn. Agron. Jour. 47:388.

Hughes, G. R. and A. L. Hooker. 1971. Gene action conditioning resistance to Northern leaf blight in maize. Crop Sci. 11:180-184.

Hursh, C. R. 1924. Morphological and physiological studies on the resistance of wheat to Puccinia graminis tritici Erikss. and Henn. Jour. Agr. Res. 27:381-411.

James, W. C. 1972. Assessing severity of plant diseases: leaf rusts of cereals. Ottawa Res. Sta. Canada Dept. of Agriculture, Res. Public. 110.

Janes, M. J. 1973. Corn earworm and fall armyworm occurrence and control on sweet corn ears in South Florida. Jour. Econ. Entomol. 66:973-974.

Jenkins, M. T. and A. L. Robert. 1959. Evaluating the breeding

potential of inbred lines of corn resistant to the leaf blight caused by Helminthosporium turcicum. Agron. Jour. 51:93-96.

Jenkins, M. T., A. L. Robert, and W. R. Findley, Jr. 1954. Recurrent selection as a method for concentrating genes for resistance to Helminthosporium turcicum leaf blight in corn. Agron. Jour. 46:89-94.

Jennings, P. H., B. L. Brannaman, and F. P. Zscheile, Jr. 1969. Peroxidase and polyphenol oxidase activity associated with Helminthosporium leaf spot of maize. Phytopathology 59:963-967.

Johnson, G. R. 1973. Relationship between yield and several yield components in a set of maize hybrids. Crop Sci. 13:649-651.

Jones, G. L., D. F. Matzinger, and W. K. Collins. 1960. A comparison of fluecured tobacco varieties repeated over locations and years with implications on optimum plot allocation. Agron. Jour. 52:195-199.

Josephson, L. M. and B. Naidu. 1971. Reaction in diallel crosses of corn inbreds (Zea mays L.) to maize dwarf mosaic virus. Crop Sci. 11:664-667.

Kappelman, A. J. Jr. and D. L. Thompson. 1966. Inheritance of resistance to diplodia stalk-rot in corn. Crop Sci. 6:288-290.

Klarman, W. L. and J. W. Gerdemann. 1963. Resistance of soybeans to three Phytopathora species due to the production of phytoalexin. Phytopathology 53:1317-1320.

Laundon, G. F. and J. M. Waterston. 1964. Puccinia sorghi. C. M. I. Descriptions of pathogenic fungi and bacteria. No. 3.

Lee, B. H., A. L. Hooker, W. A. Russell, J. G. Dickson, and A. L. Flangas. 1963. Genetic relationships of alleles on chromosome 10 for resistance to Puccinia sorghi in 11 corn lines. Crop Sci. 3:24-26.

LeRoux, P. M. and J. G. Dickson. 1957. Physiology, specialization, and genetics of Puccinia sorghi on corn and of Puccinia purpurea on sorghum. Phytopathology 47:101-107.

Levings, C. S., III, C. W. Stuber, and C. F. Murphy. 1971. Inheritance of an auxin inducible peroxidase in oats (Avena sativa L.). Crop Sci. 11:271-272.

Lewis, R. W. 1953. An outline of the balance hypothesis of parasitism. Amer. Naturalist 87:273-281.

Liang, G. H. L. and T. L. Walter. 1966. Genotype X environment

interactions from yield tests and their application to sorghum breeding programs. Canad. Jour. Genet. Cytol. 8:306-311.

Lim, S. M., A. L. Hooker, and J. D. Paxton. 1970. Isolation of phytoalexins from corn with monogenic resistance to Helminthosporium turcicum. Phytopathology 60:1071-1075.

Lim, S. M., J. D. Paxton, and A. L. Hooker. 1968. Phytoalexin production in corn resistant to Helminthosporium turcicum. Phytopathology 58:720-721.

Link, K. P., H. R. Angell, and J. C. Walker. 1929. The isolation of protocatechuic acid from pigmented onion scales and its significance in relation to disease resistance to onions. Jour. Biol. Chem. 81:369-375.

Link, K. P. and J. C. Walker. 1933. The isolation of catechol from pigmented onion scales and its significance in relation to disease resistance in onions. Jour. Biol. Chem. 100:379-383.

Littlewood, R. K., S. G. Carmer, and C. N. Hittle. 1964. A computer program for estimating combining abilities in relation to diallel crossing systems. Crop Sci. 4:662-663.

Loebenstein, G. and N. Linsey. 1961. Peroxidase activity in virus-infected sweet potatoes. Phytopathology 5:533-537.

_____. 1963. Effect of virus infection on peroxidase activity and C_6/C ratios (Abstract). Phytopathology 53:350.

_____. 1966. Alterations of peroxidase activity, associated with disease symptoms, in virus-infected plants. Israel Jour. Bot. 15:163-167.

Loesch, P. J. and M. S. Zuber. 1972. Inheritance of resistance to Maize Dwarf Mosaic Virus. Crop Sci. 350-352.

Lovrekovich, L., H. Lovrekovish, and M. A. Stahmann. 1968. The importance of peroxidase in the wildfire disease. Phytopathology 58:193-198.

Lowry, O. H., N. J. Rosebrough, A. L. Farr, and R. J. Randall. 1951. Protein measurement with the Folin phenol reagent. Jour. Biol. Chem. 193:265-275.

Luckmann, W. H., A. M. Rhodes, and E. V. Wann. 1964. Silk balling and other factors associated with resistance of corn to corn earworm. Jour. Eco. Entomol. 57:778-779.

MacDonald, T. and J. L. Brewbaker. 1972. Isoenzyme polymorphism in flowering plants. VIII. Control and dimeric nature of two

transaminase isoenzymes in maize. Jour. Heredity 63:11-14.

Mains, E. B. 1931. Inheritance of resistance to rust, Puccinia sorghi, in maize. Jour. Agr. Res. 43:419-430.

_____. 1962. Studies in rust resistance. Jour. Heredity 17:313-325.

Mains, E. B., F. J. Frost, and G. M. Smith. 1924. Corn resistance to rust, Puccinia sorghi (Abstract). Phytopathology 14:47-48.

Malm, N. R. and J. B. Beckett. 1962. Reactions of plants in the tribe Maydeae to Puccinia sorghi Schw. Crop Sci. 2:360-361.

Malm, N. R. and A. L. Hooker. 1962. Resistance to rust, Puccinia sorghi Schw., conditioned by recessive genes in two corn inbred lines. Crop Sci. 2:145-147.

Manandhar, K. L. and S. M. Shah. 1972. Screening of different corn germ-plasms against leaf rust disease under Kakani conditions. Eighth Inter-Asian Corn Improvement Workshop. Thai National Corn and Sorghum Program: 137-140.

Mather, K. 1949. Biometrical genetics. Dover Publications. New York.

McClelland, C. K. 1929. The relations of shuck covering to earworm attack. Agron. Jour. 21:235-236.

Miller, P. A., J. C. Williams, and H. F. Robinson. 1959. Variety X environment interactions in cotton variety tests and their implications on testing methods. Agron. Jour. 51:132-134.

Moll, R. H., D. L. Thompson, and P. H. Harvey. 1963. A quantitative genetic study of the inheritance to brown spot (Physoderma maydis) of corn. Crop Sci. 3:389-391.

Moseman, J. G. 1959. Host-pathogen interaction of the genes for resistance in Hordeum vulgare and for pathogenicity in Erysiphe graminis f. sp. hordei. Phytopathology 49:467-472.

Muller, A. S. 1953. Plant disease problems in Central America. FAO Plant Protect. Bull. 1:136-138.

Muller, K. O. 1961. The phytoalexin concept and its methodological significance. Recent Advances in Botany 1:396-400. University Toronto Press, Toronto.

Murray, M. J. 1961. Spearmint rust resistance and immunity in the genus Mentha. Crop Sci. 1:175-179.

Nattrass, R. M. 1952. Preliminary notice of the occurrence of a rust (Puccinia polysora) on maize. East African Agr. Jour. 18:39-40.

Nelson, R. R. 1972. Breeding Plants for Disease Resistance. Penn. State University Press, Penn.

Nelson, L. R. and G. E. Scott. 1973. Diallel analysis of resistance of corn (Zea mays L.) to corn stunt. Crop Sci. 13:162-164.

Nigeria, Agr. Dept. 1953. Report of the Agricultural Department; Western Provinces and Colony. Nigeria Agr. Dept. Annual Rept. 1950/51:47-86.

Novacky, A. and R. E. Hampton. 1968. Peroxidase isozymes in virus-infected plants. Phytopathology 58:301-305.

Painter, R. H. 1968. Insect Resistance in Crop Plants. The University Press of Kansas, Lawrence.

Painter, R. H. and A. M. Brunson. 1940. Differential injury within varieties, inbred lines and hybrids caused by the corn earworm Heliothis armigera (Hbn). Jour. Agr. Res. 61:81-100.

Park, D. 1956. West African maize rust research unit. Nature 178:904-905.

Peirce, L. C. and J. L. Brewbaker. 1973. Application of isozymes analysis in horticultural science. HortScience 8:17-22.

Person, C. 1959. Gene-for-gene relationships in host; parasite systems. Canad. Jour. Bot. 37:1101-1110.

_____. 1966. Genetic polymorphism in parasitic systems. Nature 212:266-267.

Person, C., D. J. Samborski, and R. Rohringer. 1962. The gene-for-gene concept. Nature 194:561-562.

Peterson, R. F., A. B. Campbell, and A. E. Hannah. 1948. A diagrammatic scale for estimating rust intensity on leaves and stems of cereals. Canad. Jour. Res. (Sect. C.) 26:496-500.

Phillips, W. J. and G. W. Barber. 1931. The value of husk protection to corn ears in limiting corn earworm injury. Va. Agr. Expt. Bul. 43:24.

Phillips, W. J. and K. M. King. 1923. The corn earworm: Its ravages on field corn and suggestions for control. USDA Farmer's Bull. 1310. 14 p.

Poole, C. F. 1935. Corn-earworm resistance in maize varieties at

Davis, California. 1934. Proc. Amer. Soc. Hort. Sci. 32:453-457.

Poole, C. F. 1937. Resistance to corn-earworm in the Charleston, South Carolina, area. Proc. Amer. Soc. Hort. Sci. 34:566-569.

_____. 1940. Corn earworm resistance and plant characters. Proc. Amer. Soc. Hort. Sci. 38:605-609.

Powers, H. R., Jr., and W. J. Sands. 1960. Genetic control of the host-parasite relationship in wheat powdery mildew. Phytopathology 50:454-457.

Pryor, A. J. 1971. Biochemical basis of rust resistance in maize. CSIRO Report 18.

Reitz, L. P. 1954. Wheat breeding and our food supply. Econ. Bot. 8:251-268.

Reyes, G. M. 1953. An epidemic outbreak of the maize rust in Eastern and Central Visayas, Philippines. Philippine Jour. Agr. 18:115-128.

Rhind, D. 1952. The rust diseases of maize. West Africa Inter-Territorial Secretariat, Dept. of Agricultural and Forestry Research, Accra. Memorandum No. 1. 16 pp.

Rhind, D., J. M. Waterston, and F. C. Deighton. 1952. Occurrence of Puccinia polysora Underw. in West Africa. Nature 169:631.

Rhoades, M. M. and V. H. Rhoades. 1939. Genetic studies with factors in the tenth chromosome of maize. Genetics 24:302-314.

Rhoades, V. H. 1935. The location of a gene for disease resistance in maize. Proc. Nat. Acad. Sci. 21:243-246.

Roane, C. W. 1950. Observations on corn diseases in Virginia from 1947 to 1950. Plant Dis. Reptr. 34:394-396.

Robert, A. L. 1962. Host ranges and races of the corn rusts. Phytopathology 52:1010-1012.

Roberts, L. M., U. J. Grant, R. Ramirez E., W. H. Hatheway, and D. L. Smith in collaboration with P. C. Mangelsdorf. 1957. Races of Maize in Colombia. National Academy of Science-National Research Council, Pub. 510, Wash. D. C.

Robinson, R. A. 1969. Disease resistance terminology. Rev. Appl. Mycol. 48:593-606.

_____. 1971. Vertical resistance. Rev. Pl. Path. 50:233-239.

Robinson, H. F., R. E. Comstock, and P. H. Harvey. 1955. Genetic variances in open-pollinated varieties of corn. Genetics 40:45-60.

Rubin, B. A., E. V. Artsikhovskaya, and T. A. Proskurnikova. 1947. Oxidative conversions of phenol and their role in the phenomena of potato resistance to Phytophthora infestans. Biochemiya 12:141-152.

Rudolph, K. and M. A. Stahmann. 1964. Interactions of peroxidases and catalases between Phaseolus vulgaris and Pseudomonas phaseolicola (Hulo blight of bean). Nature 204:474-475.

Russell, W. A. 1961. A comparison of five types of testers in evaluating the relationship of stalk rot resistance in corn inbred lines and stalk strength of the lines in hybrid combinations. Crop Sci. 1:393-397.

_____. 1965. Effect of corn leaf rust on grain yield and moisture in corn. Crop Sci. 5:95-96.

_____. 1971. Types of gene action at three gene loci in sublines of a maize inbred line. Canad. Jour. of Genetics and Cytology. XIII. 2:322-334.

Russell, W. A. and S. A. Eberhart. 1970. Effects of three gene loci in the inheritance of quantitative characters in maize. Crop Sci. 10:165-169.

Russell, W. A. and A. L. Hooker. 1959. Inheritance of resistance in corn to rust, Puccinia sorghi Schw., and genetic relationships among different sources of resistance. Agr. Jour. 51:21-24.

_____. 1962. Location of genes determining resistance to Puccinia sorghi Schw. in corn inbred lines. Crop Sci. 2:477-480.

Saxena, K. M. and A. L. Hooker. 1964. The nature of locus Rp_1 conditioning resistance to rust in corn (abstract). Phytopathology 54:905.

_____. 1968. One the structure of a gene for disease resistance in maize. Proc. Nat. Acad. USA 61:1300-1305.

Scandalios, J. G. 1969. Genetic control of multiple molecular forms of enzymes in plants: a review. Biochem. Genetics 3:37-79.

Schieber, E. and J. G. Dickson. 1963. Comparative pathology of three tropical corn rusts. Phytopathology 53:517-521.

Scott, G. E. and L. R. Nelson. 1972. Effectiveness of resistance in maize to Maize Dwarf Mosaic. Agron. Jour. 64:319-320.

Seevers, P. M. and J. M. Daly. 1970. Studies on wheat stem rust resistance controlled at the Sr6 locus. II. Peroxidase activities. Phytopathology 60:1642-1647.

Seevers, P. M., J. M. Daly, and F. F. Cathedral. 1971. Isozymes in resistance to wheat stem rust disease. Plant Physiol. 48:353-360.

Semeniuk, G. and E. F. Vestal. 1952. Leaf diseases of corn in Iowa in 1951. Plant Dis. Reptr. 36:171-177.

Semeniuk, G. and J. R. Wallin. 1947. The influence of climate on four leaf parasites of Zea mays in Guatemala (Abstract). Phytopathology 37:20.

Shieber, E. and J. G. Dickson. 1963. Comparative pathology of three tropical corn rusts. Phytopathology 53:517-521.

Shieber, E., A. E. Rodriguez V., and S. Fuentes F. 1964. Distribution of Puccinia sorghi and P. polysora in Central Mexico. Plant Dis. Reptr. 48:425-427.

Shin, Han Poong. 1972. Gene action in the inheritance of agronomic traits in intervarietal diallel crosses and relative importance of gene effects for quantitative characters in Zea mays L. Ph. D. thesis. Dept. of Agronomy, University of Hawaii, Honolulu.

Smith, G. A. and E. G. Ruppel. 1974. Heritability of resistance to cercospora leaf spot in sugarbeet. Crop Sci. 14:113-115.

Sprague, G. F. 1955. Corn breeding. In: G. F. Sprague, ed., Corn and Corn Improvement. pp. 221-292. Academic Press, N. Y.

_____. 1966. Quantitative Genetics in plant improvement. In: K. J. Frey, ed., Plant Breeding. pp. 315-354. Iowa State University Press, Ames, Iowa.

Stakman, E. C. and J. J. Christensen. 1926. Physiologic specialization of Ustilage zeae and Puccinia sorghi and their relation to corn improvement (Abstract). Phytopathology 16:84.

Stakman, E. C., J. J. Christensen, and H. E. Brewbaker. 1928. Physiologic specialization in Puccinia sorghi. Phytopathology 18:345-354.

Stakman, E. C. and J. G. Harrar. 1957. Principles of Plant Pathology. The Ronald Press Company, New York.

Starks, K. J. and W. W. McMillian. 1967. Resistance in corn to the corn earworm and fall armyworm. Part. II. Types of field resistance to the corn earworm. Jour. Econ. Entomol. 60:920-923.

Stavely, J. R. and E. W. Hanson. 1967. Electrophoretic comparisons of resistant and susceptible Trifolium pratense noninoculated and inoculated with Erisiphepolygoni. Phytopathology 57:482-485.

Stevenson, F. J. and H. A. Jones. 1953. Some sources of resistance in crop plants. Yearbook Agri. (U. S. Dept. Agr.) pp. 192-216.

Straub, R. W., M. L. Fairchild, and A. L. Keaster. 1973. Corn earworm: Use of larval traps on corn ears as a method of evaluating corn lines for resistance. Jour. Econ. Entomol. 66:989-990.

Suneson, C. A. 1960. Genetic diversity--a protection against plant diseases and insects. Agron. Jour. 52:319-321.

Thatcher, F. S. 1943. Cellular changes in relation to rust resistance. Canad. Jour. Res. (C) 21:151-172.

Timothy, D. H., W. H. Hatheway, U. J. Grant, M. Torregroza C., D. Sarria V., and D. Varela A. 1963. Races of Maize in Ecuador. National Academy of Science-National Research Council. Pub. 975. Wash. D. C.

Tomiyama, K. and M. A. Stahmann. 1964. Alternation of oxidative enzymes in potato tuber tissue by infection with Phytophthora infestans. Plant Physiol. 39:483-490.

Townsend, G. R. 1951. Control of the leaf blight and rust diseases of sweet corn. Plant Dis. Reptr. 35:368-369.

Ullstrup, A. J. 1955. Diseases of corn. In: Corn and Corn Improvement. G. F. Sprague, ed. pp. 465-536. Academic Press, N. Y.

_____. 1958. The occurrence of southern rust and other corn diseases in Indiana in 1957. Plant Dis. Reptr. 42:373.

_____. 1965. Inheritance and linkage of a gene determining resistance in maize to an American race of Puccinia polysora. Phytopathology 55:425-428.

_____. 1970. A comparison for monogenic and polygenic resistance to Helminthosporium turcicum in corn. Phytopathology 60:1597-1599.

Umaerus, V. 1959. The relationship between peroxidase activity in potato leaves and resistance to Phytophthora infestans. Amer. Potato Jour. 36:124-131.

Valle, C. G. Del and J. C. Miller. 1963. Influence of husk length and tightness against corn earworm damage in sweet corn hybrids. Amer. Soc. Hort. Sci. 83:531-535.

Van der Plank, J. E. 1968. Disease Resistance in Plants. Academic

Press, New York and London.

Van Dyke, C. G. and A. L. Hooker. 1969a. The Z reaction in corn to Puccinia sorghi. Phytopathology 59:33-36.

_____. 1969b. Ultra-structure of host and parasite in interaction of Zea mays with Puccinia sorghi. Phytopathology 59: 1934-1946.

Vestal, E. F. and G. Semeniuk. 1951. Puccinia sorghi and Helminthosporium turcicum on corn in Iowa during 1950. Plant Dis. Reptr. 35:212-214.

Waker, J. C. 1959. Progress and problems in controlling plant disease by host resistance. In: Plant Pathology, Problems and Progress 1908-1958. C. S. Hotton, G. W. Fisher, R. W. Fulton, H. Hart, and S. E. A. McCallan, eds. pp. 32-41. Univ. of Wisconsin Press, Madison, Wis.

Wallin, J. R. 1951. An epiphytotic of corn rust in the North Central Region of the United States. Plant Dis. Reptr. 35:207-214.

Walter, E. V. 1961. Source of earworm resistance for sweet corn. Amer. Soc. Hort. Sci. 80:485-487.

Warner, J. N. 1952. A method for estimating heritability. Agron. Jour. 44:424-430.

Weber, G. F. 1921. Studies on corn rust (Abstract). Phytopathology 11:31-32.

Weber, C. R. 1950. Inheritance and interrelation of some agronomic and chemical characters in an interspecific cross in soybeans. Iowa, Agr. Exp. Sta. Bull. 374:767-816.

Weber, D. J. and M. A. Stahmann. 1964. Ceratocystis infection in sweet potato: Its effect on proteins, isozymes, and acquired immunity. Science 146:929-931.

_____. 1966. Induced immunity to Ceratocystis infection in sweet potato root tissue. Phytopathology 56:1066-1070.

Weber, D. J., B. Clare, and M. A. Stahmann. 1967. Enzymic changes associated with induced and natural resistance of sweet potato to Ceratocystis fimbriata. Phytopathology 57:421-424.

Wellhausen, E. J., A. Fuentes, O., and A. H. Corzo in collaboration with P. C. Mangelsdorf. 1957. Races of Maize in Central America. National Academy of Sciences-National Research Council. Pub. 511. Wash. D. C.

Wellhausen, E. J., L. M. Roberts, and E. Hernandez X., with collaboration with P. C. Mangelsdorf. 1952. Races of Maize in Mexico: Their Origin, Characteristics and Distribution. Bussey Institution of Harvard University.

Wellensiek, S. J. 1927. The nature of resistance in Zea mays L. to Puccinia sorghi Schw. Phytopathology 17:815-825.

Widstrom, N. W. 1967. An evaluation of methods for measuring corn earworm injury. Jour. Econ. Entomol. 60:791-794.

Widstrom, N. W. and J. H. Hamm. 1969. Combining abilities and relative dominance among maize inbreeds for resistance to earworm injury. Crop Sci. 9:216-210.

Widstrom, N. W. and W. W. McMillian. 1973. Genetic effects conditioning resistance to earworm in maize. Crop Sci. 13:459-461.

Widstrom, N. W. and K. J. Starks. 1967. Influence of environment on injury to corn by the corn earworm. Jour. Econ. Entomol. 60: 181-185.

Widstrom, N. W., W. J. Wiser, and L. F. Bauman. 1970. Recurrent selection in maize for earworm resistance. Crop Sci. 10:674-676.

Widstrom, N. W., B. R. Wiseman, and W. W. McMillian. 1972. Genetic parameters for earworm injury in maize populations with Latin American germ plasm. Crop Sci. 12:358-359.

Wilkinson, D. R. and A. L. Hooker. 1968. Genetics of reaction to Puccinia sorghi in ten corn inbred lines from Africa and Europe. Phytopathology 58:605-608.

Wingard, S. A. 1953. The nature of resistance in diseases. In: U. S. Dept. Agr. Yearbook of Agr. Plant Diseases, pp. 165-173.

Wiseman, B. R., W. W. McMillian, and N. W. Widstrom. 1970. Husk and kernel resistance among maize hybrids to an insect complex. Jour. Econ. Entomol. 63:1260-1262.

Wood, J. I., and B. R. Lipscomb. 1956. Spread of P. polysora with a bibliography on the three rusts of Zea mays. USDA special Publication 9.

Wood, R. S. K. 1967. Physiological Plant Pathology. Oxford: Blackwell.

Yarnell, S. H. 1952. Breeding sweet corn for rust resistance to the corn earworm. Proc. Amer. Soc. Hort. Sci. 60:379-386.

Yarwood, C. E. and A. L. Hooker. 1966. Heat predisposition to corn